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BOSTON CITY HOSPITAL STAFF CLINICAL MEETING, OCTOBER 10, 1924

Dr. Henry Jackson, Sr.; They have given me the honor of presiding. We have tonight a very important subject, one of very great interest to the pathologist, to the clinician, and to the laboratory worker, and one in which the clinician is always looking to the laboratory for help in diagnosis, in prognosis and in treatment. Of course, there has always been a question as to which was the primary factor in the cardiorenal disease. There were two schools, one claiming that the kidney disease gave rise to the arterial, and the other claiming that the arterial disease was the cause of the kidney disease. I suppose Dr. Peabody can tell us whether that is definitely settled now; but it has been under discussion ever since my student days. In my studies with Dr. Fitz arteriocapillary fibrosis was the role on which he placed great dependence.

We have a most excellent program, starting with Dr. Wearn's talk on "Physiology of Kidney Function," and I take pleasure in introducing Dr. Wearn.

PHYSIOLOGY OF KIDNEY

BY J. T. WEARN, M.D.

Dr. Wearn spoke as follows:—When William Bowman published his observations on the structure of the kidney, and announced his theory of urine formation, he laid the foundation of a controversy which has raged ever since. All of you are doubtlessly familiar with the hypotheses of the two schools in renal physiology—secretion and filtration—and equally familiar with the great names of Ludwig, Heidenhain and the others that we associate with these schools. They and many others have contributed largely to our knowledge of kidney function. In more recent years Cushny has formulated his so-called "modern theory" which, in brief, is filtration of a protein free fluid through the glomeruli and selective re-absorption in the tubules. In this country A. N. Richards has added greatly to our knowledge of the kidney and has thrown much new light upon the function of both the glomeruli and the tubules.

Time does not permit a review of the massive evidence on renal function which has accumulated in recent years, so I shall attempt in the time that remains to tell you of some of the recent work of Richards and his co-workers at the University of Pennsylvania.

Some years ago, while studying the effect of minute doses of epinephrin and pituitrin upon the kidney, Richards observed certain phenomena which led him to believe that these drugs in minute doses might have a definite action upon the vessels going to and from the glomeruli. This, of course, gave him a desire to see the living kidney. (Shortly before this time Professor Krogh of Copenhagen had published his observations upon the capillaries in the frog's tongue.) Professor Richards, therefore, prepared a frog by exposing the kidney in such a way that it could be illuminated and observed microscopically. In observing the living kidney thus prepared, he was able to see the blood flow through the glomeruli, the clear spaces in Bowman's capsule and the larger arteries and veins —the tubules being visible only in outline. While studying this preparation, he noted that in some of the glomeruli with an active flow of blood through their capillaries the circulation suddenly ceased and the glomeruli practically disappeared from view. At the same time another glomerulus would appear in the same field in a space which before had contained none. This observation was verified many times, and by observing the glomeruli over a long period it was found that flow through them alternated, that is to say, some of them worked for a while and rested, and others came in and took up the work.

Further than this, he noted also that there was a difference in the diameters of the capillaries within the different glomeruli. In some a rapid flow through a narrow capillary was noted, while in others a slow, pulsating flow through dilated capillaries was noted, and various degrees of flow between these two types. With this preparation he next studied the effects of diuretics, together with Dr. Schmidt in his department. If, for instance, one microscopic field should be counted and fifteen glomeruli be found therein, the same field after the administration of a diuretic might contain twice that number or more. In other words, the diuretic brought into play additional units. Moreover, the individual capillaries within the glomerulus, after administration of caffeine, for instance, showed an increase in diameter, thus permitting greater blood flow through them. This increase in the number of glomeruli also followed the use of glucose, saline, and various other diuretics. This, you will realize, is a new conception of the kidney, making it a group of units few of which work at a given time, and with a great resereve to call in in case of need.

During these observations it was noted that a diuretic glomerulus, that is to say, one that is forming urine very rapidly, might show a distended capsule of Bowman, and it seemed possible that a method might be perfected for obtaining some of this fluid within the capsule for analysis. Consequently, a small quartz pipette was constructed of such diameter that it could be inserted through this capsule into the glomerular sac without injury to the capillaries or the other structures of the kidney. By arranging this pipette in such a way that suction could be applied, it was possible to obtain within a few hours as much as a milligram of glomerular fluid. This was then transferred to small glass capillary tubes about four or five times the diameter of the pipette for analysis. It was found that reagents could be added to the glomerular fluid in these capillary tubes and chemical tests carried out as in the gross test tube. Using a very sensitive test for protein, it was found in eleven samples that no protein was present in glomerular urine—direct confirmation of Cushny's theory. Bladder urine collected over the same period was found to be protein free also. Using Benedict's qualitative reagent, it was found that the glomerular urine gave a test for sugar, while bladder urine collected over the same period of time contained none. This, of course, was direct evidence of re-absorption in the tubules.

Tests were next carried out for chloride, and it was found to be present in the glomerular urine and either absent or present in very slight traces in the bladder urine.

It was possible by the use of an extremely delicate nephelometric method to quantitate the chloride in the glomerular urine. The results obtained confirmed the qualitative results, and showed the concentration of chloride in the glomerular urine to be very close to that of the blood plasma—the figure for the glomerular urine being a little higher than that of the plasma.

A quantitative test for urea was also devised by modifying Folin's method. The results, while not so accurate as in the chloride method, showed the pressure of urea in the glomerular urine, and its presence in the bladder urine in much greater concentration.

Studies were then carried on with various dyes, using in particular, indigo-carmine, methylene-blue and phenolsulphonephthalein, the last, the dye so commonly used for clinical function tests. In brief, the behavior of indigo-carmine was as follows: if the dye was injected while an observer watched the kidney under the microscope, one noted first that the afferent vessels to the glomeruli became blue, then the glomerular tuft became blue, and finally it was possible to see the dye go through the capillaries into the clear spaces of Bowman's capsule surrounding the glomeruli tuft. In other words, here one could actually see the dye going through the glomeruli capillaries.

It will be remembered that indigo-carmine was the dye used by Heidenhain in his experiments upon the rabbit kidney, and he concluded that the dye was secreted by the tubules. The finding of small granules of the dye in the lumen of the tubules and in the cells lining the lumen and the absence of dye in the glomerular capillaries led Heidenhain to this conclusion.

In the frog's kidney, however, one sees the dye passing through the glomerulus and some fifteen minutes later small granules of dye begin to appear in the tubules. It seems to me unnecessary to assume that the dye is excreted by tubule cells when we know re-absorption is taking place in the tubules.

These studies, it seems to me, give us a new picture of the kidney. We can now think of it as a large group of units, each with a definite function, and as many being called into use at any one time as is necessary to keep the urea. salt and products of metabolism at the desired level in the blood. They also throw new light upon the interpretation of the phenolsulphonephthalein test, and more particularly upon the two hour renal test. One does not have difficulty in imagining that a fixation of gravity in the two hour renal function test simply means that so many glomeruli have been destroyed that the remaining ones must work full time, putting out a maximum quantity of solids and fluid at all times, thus accounting for the fixation in volume, gravity, and the large night volume.

Further studies are being carried on in Dr. Richards' laboratory, and give promise of throwing more light on the physiology of the kidney.

CLINICAL STUDIES IN NEPHRITIS

BY W. R. OHLER, M. D.

Junior Visiting Physician at Boston City Hospital

My part of the program is to attempt to correlate what has already been said with the clinical management of the patient. In other words, with improved knowledge of renal physiology and blood chemistry can we take better care of our nephritic patients.

In attempting to take account of stock let us attack the problem from the point of view of Diagnosis, Prognosis and Treatment, and let us classify Nephritis into two broad divisions,—Acute and Chronic.

I. Diagnosis

A. *Blood Chemistry*

1. Acute Nephritis. Is there anything in blood chemistry which enables us to make a more accurate or an earlier diagnosis of Acute Nephritis? In general the answer is "no." Fortunately the ordinary laboratory methods, together with history and clinical findings, are sufficient to make the diagnosis, and such routine measures may give more reliable information. Often, for example, in Acute Nephritis the blood nitrogen is either not elevated at all, or only slightly so. There are, however, certain conditions where it is difficult to differentiate between an Acute Nephritis and an acute exacerbation of a chronic process. In a case of supposed Acute Nephritis where the blood nitrogen is persistently high, and does not respond to ordinary means, the chances are that we are dealing with an acute exacerbation of a Chronic Nephritis.

2. Chronic Nephritis. What part does blood chemistry play as an aid in the diagnosis of Chronic Nephritis? Dr. Jackson has already mentioned the fact that conditions other than Nephritis may cause an increase in the blood nitrogen, and also that even in the presence of a Chronic Nephritis the blood chemical findings may be normal. As a matter of fact it has been shown that as much as three fourths of the kidney substance may be destroyed before the residual blood nitrogen begins to go up. Furthermore, I agree with Dr. Jackson in what he says about uric acid determinations in this respect. It is true that if we can eliminate all extra-renal conditions which may cause an elevated blood nitrogen, slight and persistent increases in blood nitrogen readings probably mean kidney damage. In general, however, blood chemistry lends but little aid to the diagnosis of Chronic Nephritis, a fact which emphasizes all the more the importance of a careful history and physical examination.

Here, it may be well to mention the condition known as Nephrosis. Dr. Jackson has already explained what is meant by Nephrosis, so it is unnecessary for us to say more about it. Incidentally, recent work at the Rockefeller Institute points to the condition as a clinical entity. If this be true, blood chemical findings are of real assistance as an aid to the diagnosis of this condition.

B. *Kidney Physiology*

We know that the kidney excretes fluids rapidly and solids slowly; that the specific gravity varies with the amount of urine, the smaller the amount the higher the specific gravity and vice versa; that the night urine is usually low in amount and high in specific gravity; that in any given twenty-four hour period there is a considerable variation in both the amount of urine and specific gravity from time to time. Furthermore, Dr. Wearn has just told us some new facts about the glomeruli. We know that not all the glomeruli are functioning at the same time, some are at rest, others not, but if any added stimulus be given the kidney, in the form say of increased work, an increased number of glomeruli will begin to function. Furthermore, we know that the glomerular filtrate is normally free from protein.

The principle of this glomerular action is best illustrated in what is known as the Two-hour Test. This has already been explained by Dr. Wearn. It remains for me to say something about the directions for doing the test and the interpretation of results.

The following is the direction sheet given to patients:

TWO-HOUR TEST

1. Meal Hours

Breakfast	8
Dinner	12
Supper	5:30 or 6
Dinner (next day)	8

The heaviest meal is to come in the middle of the day. Absolutely nothing is to be taken between meals, not even water, and nothing from supper on the day of the test until breakfast the next day.

2. Diet

Eat an ordinary hearty diet, to include meat, salt, clear soup. The idea is to put the kidneys under a certain amount of strain.

3. Fluids

Drink the equivalent of 8 to 10 tumblers, and use more fluid at dinner than at the other meals. By fluids is meant to include water, tea, coffee, milk, etc. Keep an account of the amount of fluids used.

Detail for Test Day

8	A. M.	Pass urine. Need not be saved. Breakfast.
10	A. M.	Pass urine. Save all in Bottle No. 1.
12	Noon.	Pass urine. Save all in Bottle No. 2. Dinner.
2	P. M.	Pass urine. Save all in Bottle No. 3.
4	P. M.	Pass urine. Save all in Bottle No. 4.
5:30	P. M.	Supper.
6	P. M.	Pass urine. Save all in Bottle No. 5.
8	P. M.	Pass urine. Save all in Bottle No. 6.
8	A. M.	Before breakfast, pass urine. Save all in Bottle No. 7. Also in Bottle No. 7 or No. 8 save all urine passed during the night.

Note that the only urine to be thrown away is the first specimen at 8 A. M. on the day the test is started. By saving the 8 A. M. specimen the next day the 24-hour period is completed.

Just a few words concerning the interpretation of the Two-hour Test:—This can best be done by illustrating certain type reactions, as follows:

TYPE 1

Time	8–10	10–12	12–2	2–4	4–6	6–8	8–8	Output c.c.	Intake c.c.
Vol.	150 c.c.	40	145	50	210	70	375	1040	1800
Sp. Gr.	1008	1028	1014	1027	1009	1025	1022		

This is a perfectly normal test. Note that the urine specimens immediately following meals are high in amount and low in specific gravity, that the specimens coming between meals are low in amounts and high in specific gravity, and that the night urines are moderately small in amount with high specific gravity.

TYPE 2

Time	8–10	10–12	12–2	2–4	4–6	6–8	8–8	Output c.c.	Intake c.c.
Vol.	160 c.c.	200	75	125	60	105	725	1450	2000
Sp. Gr.	1016	1013	1017	1015	1019	1011	1014		

This is a case of hypertension with evident changes in the kidney. Note that the highest specific gravity is 1019 and the lowest is 1011, that there is a fair variation in specific gravity up until 6 o'clock at night, after which the specific gravity figures are definitely below what one would normally expect, and also that there is definite nocturia.

TYPE 3

Time	8–10	10–12	12–2	2–4	4–6	6–8	8–8	Output c.c.	Intake c.c.
Vol.	110 c.c.	50	125	190	160	80	500	1215	2000
Sp. Gr.	1012	1018	1015	1010	1010	1006	1006		

In the above test the specific gravity of the first two specimens is about what one would normally expect, but from noon on it will be noted that there is a distinct tendency toward a fixation of specific gravity, and the night amount is high and the specific gravity extremely low. This test illustrates very well how difficult it is to state just how much variation in specific gravity is normal. For example, if one takes the highest specific gravity 1018 and the lowest specific gravity 1006, there is a variation of 12 points. However, if the test is studied as a whole it will be noted that the power of the kidney to concentrate urine diminishes as the day advances. This test was done on an early case of Chronic Nephritis.

TYPE 4

Time	8–10	10–12	12–2	2–4	4–6	6–8	8–8	Output c.c.	Intake c.c.
Vol.	30 c.c.	45	25	25	65	50	300	545	1580
Sp. Gr.	1026	1023	1026	1026	1022	1022	1022		

Note that the specific gravities tend to be fixed at a rather high level, that there is a very poor response to fluid intake, and that the amount of urine at each two-hourly interval tends to be fixed, except that the night urine is not abnormal in amount. This is the type of reaction one is likely to get in cases of myocardial insufficiency with a certain amount of kdney congestion.

TYPE 5

Time	8–10	10–12	12–2	2–4	4–6	6–8	8–8	Output c.c.	Intake c.c.
Vol.	300 c.c.	190	150	235	165	320	610	1970	2000
Sp. Gr.	1009	1012	1009	1008	1010	1007	1011		

This test shows nearly maximum impairment. Note that the specific gravity is definitely fixed regardless of the amount of urine, that the night urine is high in amount and low in specific gravity.

The important point to emphasize concerning the Two-hour Test is that any abnormality should be investigated. Slight abnormalities in the test may mean kidney damage. I believe this because we have followed cases in the Out-Patient Department long enough to note development from very slight questionable changes to the perfectly definite changes characteristic of a chronic process. On the other hand I think it unwise to draw definite conclusions from a single test and for this reason it is always best to repeat the test.

In addition to the Two-hour Test, there are other tests in which the principles of kidney physiology are applied, such as the Dilution Concentration Test, the Urea Concentration Test of McLean, and the Phthalein Test. Recently, D. C. Hoffman, a third year Harvard Medical Student, and myself have carried on a comparative study in a group of patients, using all the above mentioned tests. We found that the Two-hour Test gave satisfactory and accurate information. Fortunately the Two-hour Test can be done at home and by patients of only average intelligence.

To return now to the outline of this discussion, we have considered the part that blood chemistry plays, as an aid to the diagnosis of both Acute and Chronic Nephritis. Of what value is improved knowledge of renal physiology as represented by the Two-hour Test in the diagnostic problem? In Acute Nephritis the test is of little value. The urine amounts are generally low and the specific gravity high. On the other hand, in Chronic Nephritis the Two-hour Test is of great value as an aid to diagnosis. This has already been pointed out in the discussion of types of test reactions. Suffice to add that very often the Two-hour Test alone gives information suggestive of chronic kidney changes.

II. PROGNOSIS

A. *Blood Chemistry*

1. Acute Nephritis. Ordinarily in Acute Nephritis the blood urea or the total non protein nitrogen falls rapidly with clinical improvement. In obstinate cases the fall in the blood nitrogen determinations is much slower but in such cases the clinical improvement is also slow. In other words, as an indication of the immediate prognosis, blood chemical findings do not add to the information which can be derived from routine laboratory procedures and clinical observation. As a matter of fact we have repeatedly seen cases of Acute Nephritis where the blood chemical findings were perfectly normal even though the ordinary clinical findings still pointed toward an acute kidney condition. Unfortunately there is nothing in the realm of blood chemistry which gives us any information as to what is going to happen later on to a case of Acute Nephritis.

2. *Chronic Nephritis.* Blood chemical determinations are a great value as an aid to prognosis in Chronic Nephritis. In general, I think it is fair to state that in any given case where with proper diet and nursing the blood urea can be kept under control, the immediate prognosis is good. On the other hand, in any given case where with proper diet and proper general treatment the blood nitrogen figures cannot be kept under control, or where the blood nitrogen continues to rise, the outlook is very grave. Incidentally, in Chronic Nephritis, blood nitrogen determinations are not always in agreement with clinical observation. Repeatedly we have seen cases where the bedside notes stated that the patient was improving despite the fact that the blood nitrogen determination showed the opposite to be the case. In all such instances the end results have always justified the laboratory findings. In other words, it is not easy to tell how chronic nephritics are getting along by clinical observation alone. Repeated laboratory tests in such cases give much more accurate information.

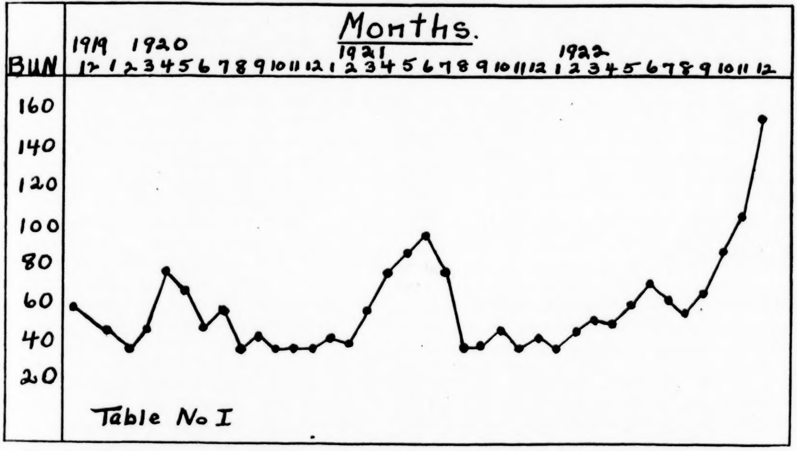

Table No I

B. *Kidney Physiology*

1. *Acute Nephritis.* During the acute stage of an acute nephritis process, the Two-hour Test gives no information insofar as prognosis is concerned. However, as the acute process subsides, we not infrequently find a definite fixation of specific gravity at a low level together with a nocturia. These findings often occur in the face of normal blood chemical values and normal phthalein. This does not indicate that the case is going to develop a chronic kidney because the ability to concentrate urine may return. When it comes to the question, however, as to what is going to happen to a case of Acute Nephritis in years to come, repeated Two-hour Tests may be of some value. For example, we have followed in the Out-Patient Department several of the so-called cured cases of Acute Nephritis for over a period of several years. In one such case we are now beginning to get slight abnormal changes in the Two-hour Test. Whether or not this indicates the onset of a chronic process, we cannot say. At any rate, it does suggest the importance of following cases of Acute Nephritis carefully over a period of years after the acute process has cleared up.

2. *Chronic Nephritis.* The Two-hour Test is of no value as an aid to prognosis in Chronic Nephritis. In other words, there may be fixation of specific gravity over a long period of years without any appreciable change in the patient's clinical condition.

III. TREATMENT

In general the treatment of Nephritis has not changed in recent years. It is still a problem of dietary control, removal of all possible foci of infection and a proper adjustment between rest and activity. The only exception to this statement is in the cases of so-called Nephrosis where some good results have been reported by giving a high protein diet.

A. *Blood Chemistry*

1. *Acute Nephritis.* In Acute Nephritis with the healing of the acute process, the blood nitrogen tends to come down to normal. In this respect we may get some idea of the effect of treatment.

2. *Chronic Nephritis.* Frequent blood deter-

minations serve as a great aid in checking up the efficacy of treatment in Chronic Nephritis. As a matter of fact, I believe that there is no other way in which one can keep such cases under control. A good example of the value of such determinations is shown in Chart No 1.

In this particular case we were able by dietary methods to keep the process under control for nearly two years. The rises in the blood urea all came as a result of acute infections and, at the time of the terminal rise, the situation was complicated by severe bronchitis. This chart indicates, therefore, not only the value of checking up the treatment of such cases by frequent blood determinations, but it also emphasizes the importance of utilizing every possible means to keep such patients free from acute infections. In the Nephritic Clinic we have felt that with the aid of frequent laboratory check-ups we have been able to outline diets more intelligently, and as a result have been able to actually prolong life.

The Two-hour Test and other similar tests are of no particular value as an aid to the treatment of either acute or chronic kidney conditions.

In closing, I would like to make a plea not necessarily for more laboratory work, but for more intelligent laboratory work and for a more careful checking up of cases of Nephritis over long periods of time.

BLOOD CHEMISTRY IN CHRONIC NEPHRITIS

BY HENRY JACKSON, JR., M.D.

THE subject of blood chemistry in chronic nephritis is far too large a field to be covered adequately in the few minutes at my disposal. It is hence necessary, perhaps at the risk of avoiding disputed points and certainly at the risk of being incomplete in certain respects, to be somewhat dogmatic. I shall make no attempt to clear up unsettled contentions nor shall I discuss new and untried fields. On the contrary I shall confine myself to what I believe to be of real importance to the practitioner of today.

The value of any blood chemical determination is directly proportional in the first place to the ease and accuracy with which the method can be done and it is proportional in the second place to the fidelity and uniformity with which definite quantitative changes in the blood constituent in question parallels more or less clearly defined clinical entities or clinical states of the same entity. Proper chemical analyses of the blood require a good laboratory and a good man in the laboratory. Accurate and intelligent interpretation of the figures is essential and there must be a full realization on the part of the interpreter of the errors which may, and often do, creep in. The practitioner who demands chemical data should be familiar with the normal values and be willing and able to draw his own conclusions from the results reported to him.

For our purposes we may divide the chemical constituents of the blood into three main classes: the proteins, the simple inorganic substances and the simple organic substances, and I shall consider each of these three classes as they bear on the problem of chronic nephritis.

In the first place the proteins. The total protein content of the blood plasma,—we do not deal here of course with the whole blood,— is from 6.5 to 7.5 grams per 100 mils. The proper determination of the plasma proteins is a difficult process, one, indeed, which should be undertaken only by those thoroughly familiar with its pitfalls. Now there are certain classes of nephritis in which there are very definite changes in the protein content of the blood. In the so-called nephrosis, (characterized clinically by massive edema and gross albuminuria,) and to a less extent in what Volhard and Fahr have classified as the nephrotic type of glomerular nephritis, there is a marked reduction of the total plasma protein and this reduction is not dependant, as some have thought, on a dilution of the blood coincident with the general anasarca. There is a true and absolute reduction of the total plasma protein. This may be reduced to 3 or 4 grams per 100 mils instead of the normal 6 to 8. There is, however, no constant parallelism between the presence or absence of albuminuria and edema and the protein content of the plasma. You will recollect that Epstein and his followers have, on the basis of this theoretical finding, maintained that the edema and albuminuria in nephrosis are due to the low plasma proteins and advocated high protein feeding in these cases with a view to restoring the proteins and raising the osmotic pressure. Very striking clinical improvement has followed this procedure in some cases. In many it has failed. It is inadvisable in my opinion for the practitioner of general medicine to follow this advice unless his cases are thoroughly controlled in a large hospital and adequate means are at hand to follow the case both clinically and chemically. Much interest attaches to this lowered plasma protein, but for the present its importance is more theoretical than practical.

When we come to the consideration of the simple organic substances in chronic nephritis we deal almost entirely with the nitrogenous compounds. The first of these, the total non-protein nitrogen, amounts to from 25 to 40 mgms per 100 mils. The determination is very simple. It is the easiest, the shortest and the most reliable of the blood chemical determinations. With reasonable care it is virtually fool proof and I strongly advise it as a routine determination. For all practical purposes I think it is fair to say that the N. P. N. gives about all the information that any determination of the simple nitrogenous compounds can give. If it is normal it does not rule out nephritis; if it is high the patient has not necessarily got Bright's disease. It may be high in a number of other conditions, but if these are ruled out, a high N. P. N. means nephritis and furthermore involvement of considerable severity.

For practical purposes the blood urea nitrogen parallels the non-protein nitrogen. It is normally 35 to 55% of the total; but as latter rises the percentage increase of the urea N increases so that in cases with very great retention the urea may form as much as 75% of the total. In competent hands the determination of urea in the blood is comparatively simple, but it is by no means as reliable as many physicians and technicians think and in inexperienced hands it may prove extremely treacherous. There are many pitfalls all along the analytical route which are not obvious at the time. For practical purposes the NPN determination is far better.

The uric acid is at present on the high wave of popularity. It is determined on the slightest excuse and many claims are made as to its value in the diagnosis and prognosis of renal disease. The determination has been modified a number of times over a period of years since the first colorimetric, microchemical method of Folin and Dennis in 1913 and it is important to remember that the normal figure for blood uric acid has steadily risen with each improvement of the method. To interpret the figures at hand it is therefore of the utmost importance to know by which method the determination has been made. If it is done by Folin's 1913 method the normal may perhaps be taken as 2.5 mgm uric acid per 100 mils, if by Folin's or Benedict's latest modification (direct reaction with cyanide and heating) 4.5 mgm per 100 mils would not be excessive. Furthermore, the amount in the plasma seems to be much greater, relatively, that that in the whole blood, and this fact must be considered if the determination be done on plasma. Many observers have maintained that uric acid is the first substance to be retained in the blood and that, therefore, blood uric acid determinations promised an earlier diagnosis of renal disease than any other. As Dr. Folin has pointed out, however, the percentage increase of uric acid is seldom greater than that of the other nitrogenous constituents. There are many cases of frank nephritis in which the uric acid is raised but slightly or not at all and there are many cases in which the uric acid is raised which show no renal lesion. Schamberg has recently shown that about one-half of his eczema cases have an abnormally high uric acid; and it is very unlikely that so large a percent should also have Brights disease. Feinblatt in a series of 1500 of unselected cases found only 47 in which the uric acid was raised when the urea nitrogen was not also increased. Nor were all of these 47 cases of nephritis. It has also been recently shown that the blood uric acid may rise to very considerable heights during starvation. I have gone somewhat into detail in this question of uric acid because it has become the custom in some quarters to look upon this determination as the best indicator of the presence or absence of renal disease and I cannot but feel that it is dangerous to lay too much stress on this particular factor. The whole problem is still under consideration but the fact remains that the uric acid is raised in many conditions other than Brights disease and may well be normal or very nearly so in advanced and even moribund cases of this malady.

The amino acid nitrogen normally varies from 6 to 8 mgm per 100 mils. For practical purposes the determination is of no value in nephritis. The normal deaminization process is very complete and has a large margin of safety and though some observers have found a significant rise of this fraction in cases with nitrogen retention, the practitioner need not, for the present, concern himself with this determination.

Blood creatinine is of some importance, especially in prognosis. In health it is remarkably constant, ranging from 1.2 to 1.5 mgm per 100 mils whole blood. It is probably true that a markedly raised creatinine in the blood indicates a bad prognosis and certainly if the figure is over 5 the outlook is very grave indeed. More than this cannot be said. I have seen cases with practically a normal creatinine die in a few weeks and cases with a high creatinine live for over a year. On the whole, however, creatinine is regarded quite justifiably as determination of prognostic value.

The nucleotide nitrogen, very recently discovered, has no practical significance as far as is known.

The important simple inorganic substances are comparatively few in number. The blood phosphate runs roughly parallel to the N. P. N. The blood calcium tends to be inversely proportional to the phosphates in nephritis. A high phosphate and a low calcium is supposed to be a poor prognostic sign. English writers have especially stressed this point.

A word about the ammonia in the blood. The determination shows signs of becoming popular and this is unfortunate for the blood ammonia determination is by far the most difficult in the realm of blood chemistry and the exquisite care and technique are required for accurate results. Benedict has recently brought forward evidence that ammonia is formed in the kidney and Robert Loeb and his associates have presented data which support this theory. But at present the entire problem of blood ammonia is in the experimental stage and though extremely interesting from the scientific point of view has but little importance to the practitioner.

I may appear to have been somewhat iconoclastic. I certainly have added no new material. I have purposely avoided those matters which though they have great scientific interest have at present but little practical importance. Certain it is that the non-protein nitrogen determination is of value in the diagnosis and the treatment of renal disease, and particularly if the determination is repeated from time to time over a period of weeks and months. A steadily rising N. P. N. in the face of rational treatment is indicative of a severe and advancing disease. Blood chemical examinations may indicate that the patient is doing badly when clinical evidence to this effect may be lacking. By the same token such examinations may fail utterly. Remember that negative evidence is treacherous. The creatinine may indicate a severe prognosis. But it is still true that careful clinical observation is very important and it is well to remember that that great clinician, John Bright, early in the 19th century noted the two features which stand out so prominently today, namely, the decreased plasma protein in the large white kidney and the increased blood urea in chronic interstitial nephritis of an advanced degree.

Dr. Henry Jackson, Sr.: I think it is due to Dr. Wearn for me to tell the gentlemen here that a large percentage of the difficulties in laboratory work here were overcome by Dr. Wearn. He certainly offered a number of points which were suggestive to the clinicians and pathologists.

ORIGINAL ARTICLES

WHOOPING COUGH

Its Bacteriology, Diagnosis, Prevention and Treatment*

BY THORVALD MADSEN

Director of the Danish Serotherapeutic Institute, Copenhagen

It will perhaps surprise you that at a time when the world of medicine is up in arms to fight the great epidemic diseases which the war has sown broadcast, I have chosen as subject for the lecture which I have the honor of giving this evening such an everyday malady as whooping cough; which many doctors and laymen consider a comparatively negligible children's disease. However, the fact remains,—and not least in those countries which have been spared the great epidemics,—that whooping cough is beginning to occupy the front rank among those diseases which are attracting the attention of public health administrators.

This will perhaps sound strangely to ears accustomed to hearing of diphtheria, cerebrospinal meningitis and typhoid fever. However, figures tell another story. Let us consider for a moment conditions in my own native country, Denmark. We see from a chart (curve 1) showing the morbidity and mortality of whooping cough in Denmark from 1890 to 1922, that the disease appears with unvarying intensity year by year. If it seems on the increase, it is only because notifications of the disease are more complete of late years. You will observe that the curve of mortality closely follows the curve of morbidity, but, there is no doubt that the trend in the thirty-year period which we are considering is on the decline,—the number of deaths per 100,000 inhabitants from 1890-99, was 38.2; from 1900-09, 29.3; from 1910-19, 19.4—a decrease in mortality of about 50%.

The continued intensity of whooping cough, blazing up with renewed vigor every third or fourth year, contrasts sharply with other hitherto dreaded infectious diseases, whose causes we now know, and against which systematic measures of prevention can be taken,—diphtheria for instance, which, with the exception of the severe attack in 1920, has declined steadily since the great epidemic of the 90's. It is gratifying to note that thanks to intensive treatment the increased number of cases is attended by only a very slight increase in mortality: it is at present about 1 per 10,000. In typhoid fever the continued decrease is even more marked. We may truthfully say that that disease is moribund, for at the present moment in Denmark in a population of 3,300,000, only 400 to 500 cases of typhoid are reported annually, with but 30 to 40 deaths.

As Denmark, with its limited area, has excellent medical service and compulsory notification of the majority of epidemic diseases, quite reliable information is furnished on the processes of these epidemics and we may surely take it for granted that while the infectious diseases just mentioned, diphtheria and typhoid fever, are on the decline, this is not the case with whooping cough.

Comparing the deaths from whooping cough, that is to say, deaths from bronchial pneumonia and acute complications contracted *during* the attack of whooping cough, but not subsequently, with deaths from a series of other infectious diseases, we observe a very interesting state of affairs as the following table indicates:

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*The Cutter Lecture in Preventive Medicine, given at the Harvard Medical School, October 14, 1924.

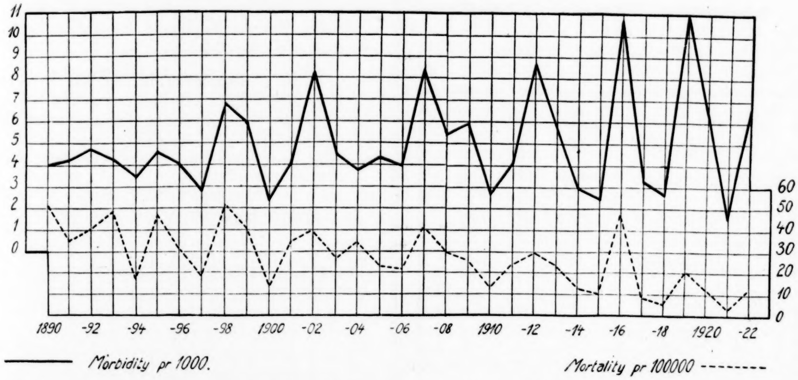
Percentage of Deaths from All Causes

	Denmark	England and Wales	U. S. Registration Area
Whooping cough	1.4%	1.3%	0.7%
Diphtheria	0.7%	0.9%	1.2%
Measles	0.5%	1.9%	0.6%
Scarlet fever	0.4%	0.3%	0.4%
Typhoid fever	0.2%	0.3%	1.0%

Whooping cough, then, in Denmark, records a far greater number of deaths than any of the other infectious diseases,—typhoid fever, measles, scarlatina, diphtheria—not to mention the more malignant, but numerically speaking far more rare epidemic diseases, such as cerebrospinal meningitis, poliomyelitis, etc. In England and Wales it is only second to measles. In the U. S. Registration Area, whooping cough has only third rank, but when you have stamped out diphtheria and typhoid fever by the admirable systematic campaign you are carrying out now, whooping cough will take the lead in the infectious children's diseases common in our climate, and for that reason demands the attention of public health officers.

CURVE 1

WHOOPING COUGH in DENMARK 1890-1922.



In order to gain particulars about the conditions in the United States, I have consulted Professor Vaughan's most excellent work, and applied to Dr. Eichel, who, at present, is in charge of the Bureau of Statistics of the Health Department of the League of Nations, and who has given me much information, of which I submit an extract in Curve 2.

Curve 2 indicates the mortality of whooping cough in the United States Registration Area for the years 1900-1921. During this period the highest rate, recorded in 1918, was 16.9, and the lowest 5.5, in the following year, 1919.

It is worth while noting that during that series of years the mortality in the United States shows no tendency to decrease, in contrast to what corresponding figures show to be the case in Denmark.

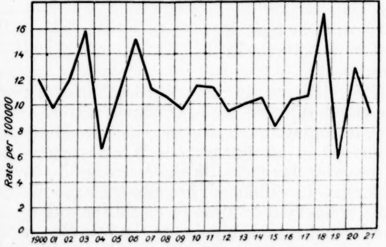
If we examine the *death rate by age*, we find that it is highest under one year, and if the life span is taken by five-year groups, it is highest in the group under 5.

This corresponds to what has been observed elsewhere. For example, let me present some statistics from Copenhagen, according to which the lethality

from 0 to 1 year was 24.3%
1 to 5 years was 2.6%
5 to 15 years was 0.7%
over 15 years was 0%

It is interesting to note how the mortality is further divided in the most threatened age group, 0-5 years. In Denmark, in the years 1910-19, per 100,000 live-born children

CURVE 2



8 died during the 1st month—circa 8 per month
75 died during the 2nd to 3rd month—circa 37 per month
424 died during the 3rd to 12th month—circa 47 per month
302 died in the 1st to 5th year
Total deaths from birth to the 5th year, 809

This goes to prove the old saying about the relative immunity, whether congenital, or due to other causes, of the first month, and the dangers of the second half of the first year.

It is interesting, too, to note how mortality varies for the *sexes.*

During the period from 1900-1920, inclusive, the death rate in the registration states of 1900 is:

	Males	Females
1900	11.1	13.4
1910	9.5	12.0
1920	10.9	13.1

In the United States, Luttinger, among others, has made this question a matter for special study and found that of 8520 cases, 44.8% were among boys, 55.2% among girls.

WHOOPING COUGH in DENMARK.

Mortality pr 100000.

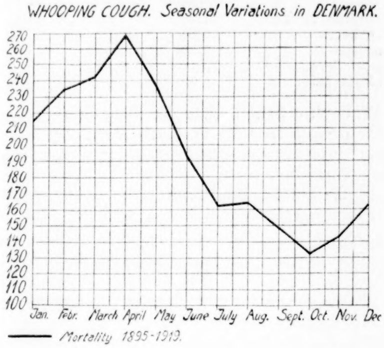
Age	Years	Males	Females
0-5	1890-1899	269	326
	1900-1909	231	269
	1909-1919	177	184
5-10	1890-1899	72	118
	1900-1909	39	90
	1909 1919	15	52

In Denmark too, a distinct difference in mortality, due to whooping cough, is observed in the two sexes, it being greater among girls than among boys. This difference has not been noted in Denmark in measles, scarlet fever or diphtheria.[*] There is a very distinct difference, then, in this age group. There is, too, every reason for emphasizing the phenomenon which coincides with the records made in Berlin, 1881-1900, and in Christiania 1880-1904, for many scientists deny it.

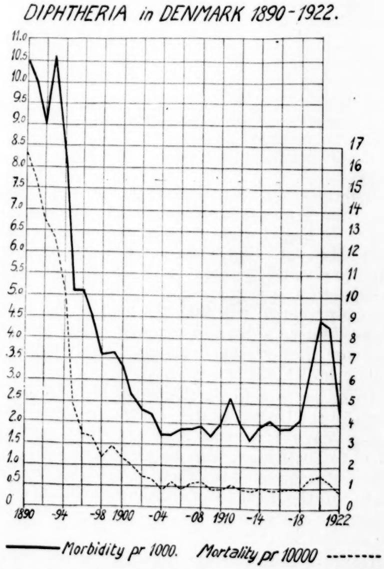
This difference in mortality in the sexes during childhood must not be confused with the fact that whooping cough is more wide-spread among women than among men; it is not permissible to compare this fact with the recently discovered difference in childhood, because the same difference is observed in measles, scarlatina and diphtheria, and is probably due to the fact that women come into more intimate contact with sick children than men, and are therefore more easily infected.

Curve 3 shows that in Denmark in the case of *seasonal variations* in pertussis, the mortality is at a pronounced minimum in September-October, increasing during the winter months until a maximum is reached in April. This coincides with a similar curve for respiratory diseases, membranous croup, bronchitis, etc. The fact is noteworthy especially as Vaughan, Luttinger and other American scientists claim that when the curve for whooping cough is at its highest, the curve for other respiratory diseases is lowest.

CURVE 3



CURVE 4



Interesting, too, is the question as to whether *whooping cough has changed in character* during the past decennial and appears now in a milder form, as the case seems to be here in Denmark with scarlatina.

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[*] H. I. Hansen: Apercu des causes de décès dans la population urbaine du royaume de Danemark, 1910-1919.

The figures given show that whooping cough is milder in form; the mortality of the last decennial is only half as great as it was in the 00's of the last century.

If the question of case fatality in Denmark is studied, we find that the case mortality is constantly on the decrease—from 6% at the end of the last century, to figures ranging about 2% now.

As is known, in former days, whooping cough often assumed a more serious character than now.

Since Guillaume Baillou described the first thoroughly substantiated whooping cough epidemic in Paris in 1578, many malignant epidemics have harrowed the various countries of both Europe and North America. Many are the lives that have been sacrificed. In the five years from 1875-80, almost 85,000 died of whooping cough in Prussia alone; in England and Wales, from 1858-65 more than 120,000. The annual death rate in the United States registration area from whooping cough for the years 1912 to 1921, inclusive, was 10.25 per 100,000 population.

When to these figures we add the fact that whooping cough may be accompanied by serious pulmonary complications, and that it may arouse a latent tuberculosis, we may perhaps realize how large a number of catastrophes may be credited to the account of this disease. There is, then, every possible reason to carry on a systematic campaign to control whooping cough.

As is the case with all infectious diseases the hygienist first enters into possession of the necessary tools *when the infectious matter* is known.

You recall, of course, that the whooping cough bacillus was first described by Bordet and Gengou in 1906 in a paper now become famous. However, as early as 1900, Bordet had observed the microbe in the expectoration of his five-month old daughter, but he was unable at that time to cultivate it.

The observations made by these two scientists have been further substantiated in a long series of articles published during subsequent years by Klimenko, C. Fränkel, Arnheim, Seiffert, Bäcker and Menschikoff, Inaba, Porcelli and many others.

Especially interesting was the discovery made by Mallory and Horner that in whooping cough there are masses of the Bordet bacillus growing in the cilia of the bronchi and the trachea of patients who have died of whooping cough.

Nevertheless, much time elapsed before the Bordet bacillus was generally acknowledged to be the cause of whooping cough. If we glance through the text books from that time,—1913,—when whooping cough investigations were commenced on a large scale at the Danish Institute of Serology, we note that doubt of its etiological importance is often expressed. Thus Jochman, the leader of the contagious ward of the Virchow Hospital in Berlin, writes in 1914 in the latest edition of his well-known text book on infectious diseases that the question of the etiology of whooping cough is, in his opinion, a problem still to be solved.

Many investigations have been made at our Danish Institute of Serology,—I name in particular the work done by Adolph Meyer and Mrs. Chievitz,—on the occurrence of the Bordet bacillus in patients and its diagnostic importance.

The first series of investigations were made using expectoration containing as little sputum as possible from the throat and mouth. Material was obtained by holding the patient's tongue down with a spatula, thus causing a paroxysm of coughing. The expectoration at the close was taken, using a sterile injection needle wound round with cotton. The expectoration was carefully washed in sterile physiological salt solution and spread out on the potato-glycerine blood agar described by Bordet and Gengou.

This method enabled the two investigators to find the Bordet bacillus in a great number of whooping cough patients, the greatest number at the beginning of the disease, and decreasing in the course of its development. The question of establishing a bacteriological diagnosis of whooping cough at our institute was now considered.

The method of procedure here described presented various practical difficulties. Taking expectoration samples was in itself no easy matter, particularly in the case of infants where an early diagnosis is especially important. A doctor is necessary or some experienced person not always easy to find. It is often difficult to avoid mixing the secretions from mouth and throat. Sometimes the patient vomits and much patience is required to obtain the expectoration necessary for the investigation.

It was a considerable step forward when Meyer and Chievitz introduced the method of allowing their patients to spit directly on the plates, the so-called cough-sowing method.

The *medium* used is practically the original medium of Bordet and Gengou, modified in a few points to lighten the work.

500 gm. sliced potatoes are boiled in 1 liter of water with 40 cm^3 glycerine until the slices become pulp. As the water boils away, more is added. The mass is strained through a sieve and wrung through a cloth. To one portion of the juice obtained, 3 parts 0.6% salt water are added. The liquid is rendered slightly alkaline (pH=7-7-5) and placed in retorts containing 200 cm^3. 8 gm agar is added to each retort. After standing covered for one night the agar is sterilized in an autoclave. It is not filtered.

If plates are to be made, equal parts agar (melted and the grease removed to 45%) and defibrinated horse blood are used. Up to 400 cm^3

blood agar mixture and 3 cm^3 normal lactic acid are added and the mixture poured into aluminium bowls 8 cm. in diameter. The bowls are made specially, they are about 1 cm. deep, the bottom, and the conjunction between bottom and sides, are grooved so that the medium cannot slip out.

If the medium is correctly made, it is of so solid a consistency that when the bowls are wrapped in a few layers of paper they can be sent by ordinary letter post without the medium's suffering injury.

The medium may be kept a fortnight on ice but only a few days at ordinary room-temperature.

The sampling is generally done by relatives of the patient. When an attack of coughing occurs the lid is removed from the bowl and for $\frac{1}{4}$ minute the bowl is held about 10 cm away from the patient's mouth, so that he coughs towards the surface of the agar. The patient must not spit on it. The bowl is returned as soon as possible to the laboratory. Here it is placed in a thermostat, where, if the patient is suffering from whooping cough, after two days, among a variegated collection of all kinds of bacteria colonies, the small, mother-of-pearl colored colonies of the Bordet-Gengou bacillus will appear. In the early stages of the disease the plate may be entirely covered, but later it is difficult to find among the myriad of other microbes. However, a little practice enables the investigator to find it with such a degree of certainty that a macroscopic test could suffice. Nevertheless the microscope is always used, and after coloring, using Gram's method and a secondary coloring with dilute carbol-fuchsine, the characteristic bacteria are found.

It is important to remember that whooping cough bacteria colonies do not always have the characteristic appearance described; the mother-of-pearl lustre is not always pronounced, and sometimes colonies are found, very white in color and shiftable on the substratum, so that they forcibly suggest Micrococcus catarrhalis. This is often the case when the media are poor and somewhat dry.

These different colony forms are not due to differences in the whooping cough strains, but to the medium alone. This is proved by the fact that when two colonies of very different appearance are sown on a new plate they will always be identical in appearance.

Should the macroscopic or microscopic appearance of the colonies on the cough plates be insufficiently typical to ensure a reliable diagnosis, it is, in our opinion, best to sow some of the culture on a new plate for comparison with a proven whooping cough bacillus. The two cultures are either identical and the case may be clearly diagnosed, "whooping cough," or they are obviously different and the case is not whooping cough. No other bacterial form has yet been found which, in a pure culture on blood agar, resembles the whooping cough bacillus and which further investigations have proved to be different from that bacillus.

One of the difficulties in the practical application of this method lies the fact that the diagnosis cannot be made earlier than two days after sowing. Sometimes three days elapse, and very rarely four, before the colony attains sufficient size to determine the species of the microbe. If practical hygienic measures are to be taken on a basis of the bacteriological diagnosis, it is of course a great disadvantage that so much time must elapse. Many experiments with quicker methods have been made at our Serum Institute, but hitherto they have been unsuccessful.

At present we are testing the possibilities in using various metal salts as medium. The question of the influence of metal salts on the growth of bacteria has been the object of many investigations made by Walbum and his collaborators at our Institute. They have discovered, for instance, that manganous chloride has an inciting effect on the growth and toxin formation of the diphtheria bacillus. However, it is by no means a matter of indifference which salt-*concentration* is used;—quite the contrary. Only a very particular concentration ensures the optimum of incitation, and if the concentration exceeds this, the salt acts as a check; if the concentration is too weak, no effect is observed. The entire question is very complicated; the optimal concentration seems to differ for each microbe and each metal salt. At present one of my assistants, Dr. K. Jensen, is testing the question with a series of bacteria and a series of different metal salts. He has succeeded in finding a decidedly incitive effect of manganous chloride on the development of tuberculosis bacilli in the Petroff medium, but he has not yet found a metal salt admixture to the whooping cough medium capable of making the bacilli grow more quickly.

We continue to hope, however, that by this means or by another we will be able to accelerate the diagnosis of whooping cough.

WHOOPING COUGH DIAGNOSIS STATIONS

We have discovered at our Institute, through the work done first by Chievitz and Meyer and later by Martin Kristensen, that during the first week of the disease under the catarrhal phase, the first week of the spasmodic phase being really the second week of the disease, the Bordet bacillus may be found in the majority of cases, 75-100 per cent. In the following week the percentage is somewhat smaller and decreases gradually from that time on, so that those patients who have had the characteristic spasmodic cough for five weeks no longer have the bacilli.

This observation, which agrees with what other scientists have observed, is of the greatest importance epidemiologically, for it confirms the observations of experienced clinicians that the contagion is greatest during the early stages of the disease before it is possible to make a clinical diagnosis with any degree of certainty. On the other hand, the fact that after 5 to 6 weeks practically no bacilli are to be found, agrees with the observations made that after that length of time the patients no longer spread contagion. In the meantime, there has been much doubt as to whether the boundary for contagion should be placed at that time. However, the above statement is backed up by a series of well-established observations made at clinics and children's hospitals, and the bacteriological observation furnishes valuable support as well. School children were formerly kept at home from school during the 3 months the spasmodic phase lasts; now in Denmark they are permitted to attend school when the spasmodic cough has lasted 4 weeks. As you will soon see, there are a few patients who, at this stage still house bacilli, but to a very limited extent. Our new regulation has proved efficient, for the schools have not reported new cases of whooping cough caught from these whooping cough convalescents.

I have tried to express the curve for bacilli secretion, but I am not yet satisfied with the form. In the first report of Chievitz and Meyer it seemed as though the curve of disappearance of the bacilli in patients approached a hyperbole which agrees with what has been observed in other infectious diseases,—for instance the disappearance of diphtheria bacilli from the throat of patients suffering from diphtheria. However, a large number of investigations made at our whooping cough diagnosis stations do not seem to corroborate this entirely as the following table indicates:

In 914 patients there were in the

catarrhal phase		75%	bacilli
spasmodic phase	1st week	57%	bacilli
	2nd week	61%	bacilli
	3rd week	45%	bacilli
	4th week	40.5%	bacilli
	5th week and later	9%	bacilli

These figures show that the bacilli remain in considerable quantities for quite a long time and then disappear comparatively quickly in the 5th and 6th weeks. It would be desirable, using a selected patient-material, to make systematic investigations of the curve of disappearance of the Bordet bacillus in order to ascertain whether it really differs so greatly from the corresponding diphtheria bactera curve as the above figures seem to indicate.

We see, then, that it is possible to make an efficient and practical whooping cough diagnosis, and the Danish doctors have done so. There are many cases in which it is very important to know whether it is a case of whooping cough or not—especially in the early stages,—and both doctors and relatives are far more apt to isolate a coughing child from other children, or an infant from possibly whooping cough-infected brothers and sisters, if there is the chance of a bacteriological diagnosis being able to shed light on the nature of the disease in a short time, than as if they were obliged to wait for a further development of clinical symptoms to give the required certainty. On the other hand, it is of practical importance to know that isolation need not be as long as was the case formerly. There is a great difference between isolating a child for 4-5 weeks and isolating it for 3 months, or as long as the characteristic whoop lasts.

It is also important to know with certainty that *carriers* after whooping cough are few even though in a single case the bacilli have been found in the 7th and 8th weeks. This is so great an exception that it may be disregarded in practice.

In this connection it is of interest to investigate the question of healthy carriers of whooping cough, a question of the greatest possible practical importance in a long series of other infectious diseases, such as diphtheria, typhoid fever, meninigitis, etc.

Danish scientists have studied the question with much interest and have carefully examined many people who have been in contact with whooping cough patients—nurses in the whooping cough wards for instance,—but they were unable to find the Bordet bacillus in any of them. It is therefore hardly right to suppose that healthy carriers play a large part in the spread of whooping cough, but, on the other hand, there is a possibility that *abortively* infected patients, in whom the disease only reaches the catarrhal stage without assuming the characteristic whooping cough form, act as carriers for the disease.

Bordet and Gengou's original description of the bacteriology of the whooping cough microbe was so exhaustive that subsequent students of the question have had little to add. That, in spite of this, so much time has elapsed before the etiological importance of the Bordet bacillus was generally accepted, is due, in large part, to the fact that it is no easy matter to distinguish it from the hæmoglobinophilic microbes, especially the Pfeiffer bacillus; there are many scientists, Belfanti and Andrews, for instance, who will not acknowledge the difference. Andrews writes, "Many bacteriologists would agree that we may be confounding under the common term, B. influenzæ, a number of closely related species. Bordet's 'whooping cough bacillus' would fall into such a group. Very little serious systematic work has so far been attempted on this group of minute hæmophilic bacilli."

In the meantime most scientists realize that two quite different microbes are meant. This is made very plain in a lengthy article by Dr. Martin Kristensen, leader of one of the departments of the State Institute of Serology. The difference between the microbes may be shown schematically in the following.

DIFFERENCES BETWEEN BORDET'S BACILLUS AND PFEIFFER'S BACILLUS

The facts stated here are the result of the investigation of at least 75 strains. Most of the facts given are to be found in the earliest literature on the whooping cough bacillus. However, Dr. Kristensen has studied them more systematically than has been done hitherto.

BORDET'S BACILLUS	PFEIFFER'S BACILLUS
Occurrence	
As far as known, only in whooping cough.	In many different diseases and in healthy persons.
Morphology	
Fairly regularly ovoid (elliptical). The bacilli may be more or less oblong, but as a rule have the same form in the entire preparation. Quite frequently decided staff-forms are also observed; these are linked in a short chain of ovoid bacilli.	In most cases more polymorphous than Bordet's bacillus; almost never shows the ovoid form characteristic of Bordet's bacillus.
Growth on Blood Agar	
Common or potato-glycerine blood agar	
(a) *Rate of Growth*	
Slow. The colonies are never macroscopically visible after 24 hours. Very tiny after 2 days; about 1 mm. in diameter after 3 days.	Quick. 1-2 mm.[3] large colonies after 24 hours.
(b) *Appearance of the Colonies*	
Either arched, resembling more or less drops of quicksilver or very white, in which case often very flat.	Flat, transparent.
(c) ***Hemolysis***	
Quite widespread, boundary indistinctly marked. Occurs 2nd-3rd day.	None.
Growth on Ordinary Agar	
Newly isolated strains do not grow, but can be made to do so later.	Can under no conditions grow on ordinary agar in pure culture.
Growth on (haemoglobine-free) Ascitic Agar	
Excellent, even in the case of recently isolated strains.	As a rule no growth, even in the case of old strains. By exception, perhaps, *puny* growth.
Growth on such media as are especially adapted to Pfeiffer's Bacillus (Fildes-Agar, Levinthal-Agar and others)	
As on common agar, the haemoglobine has no particular effect on the growth. This is true of all the Danish strains tested. One of the cultures sent from America is an exception, for it, like Pfeiffer's bacillus, grows well on Fildes and Levinthal Agar.	Very lively growth.
Symbiosis Reaction	
No.	Decided.
Viability of the Culture	
Considerable. The culture never dies during a month's cold storage.	Slight. The culture always dies during a month's cold storage.

To the above may be added some facts which Stillman and Bourn (*J. Ex. Med.*, 32, 665) and other American investigators have observed (but which we have not yet corroborated as applied to the whooping cough bacillus):

Formation of Indol	
Wanting.	Some strains positive, others negative.
Power of Fermentation	
Wanting.	Causes glycose, galactose, laevitose and in part maltose, saccharose and dextrin to ferment.

As Krumwiede, Mischulow and Oldenbusch have emphasized, the question of the various *types* of whooping cough bacillus is a matter of practical importance. These two scientists claim the existence of at least two types, A and B. The difference is based on combined agglutination and absorption experiments.

A Japanese scientist, Hayano, lays claim to four groups partly on a basis of ability to agglutinize, and partly to ferment.

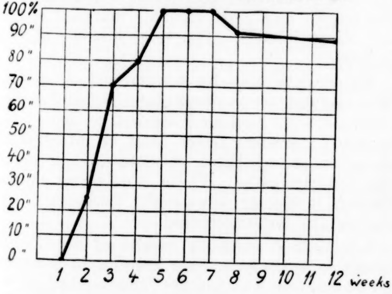
In Denmark Dr. Kristensen has investigated the question of types of whooping cough bacilli on 101 strains, but has not yet found safe ground for a clear type division. Dr. Krumwiede has been kind enough to give us his types and Dr. Kristensen is now trying to see whether there are Danish types which correspond to the American. The question of types which is important enough in the case of several other microbes must remain unsolved for the present. It is possible that the same difficulty is to be encountered here as in the case of other microbes,—pneumococci excepted,—that on reaching a certain point differences gradually disappear, so that types which seemed distinct enough begin to lose their particular signs and merge into each other. We have observed this recently in the case of the meningococci.

But is the Bordet-Gengou bacillus the real cause of whooping cough? No absolute proof has been found, but the fact that it is always present at the beginning of the disease when the infection is greatest and that its frequency decreases during the course of the disease, in exact agreement with the decline of the infection, seems to indicate that it is. Interesting, too, is the fact that it has not been observed in any great extent in healthy persons even though the possibility that a larger series of investigations based particularly on the Bordet bacillus might give another result cannot be ignored. At this very point it differs characteristically from the microbe which was formerly confused with it,—the Pfeiffer bacillus.

Another reason why it is permissible to consider the Bordet bacillus as the cause of whooping cough is the regularity with which the *complement fixation* appears in the patients. When an emulsion of Bordet-Gengou bacilli is used as antigen, the blood of whooping cough patients shows complement fixation in a majority of cases. Out of 152 blood tests on whooping cough patients, Chievitz and Meyer found 104 positive. The way in which complement fixation appears during the course of the disease is also characteristic. In the beginning of the catarrhal phase no reaction is observed—at least none has been noted at our Institute,—but after the first week the reaction increases gradually and reaches its maximum the 5th-7th weeks, after which it slowly begins to decline. Its course may be seen in Curve No. 5 and the reaction lasts for many months, sometimes a year.

CURVE 5

COMPLEMENT FIXATION REACTION
in
WHOOPING COUGH PATIENTS.



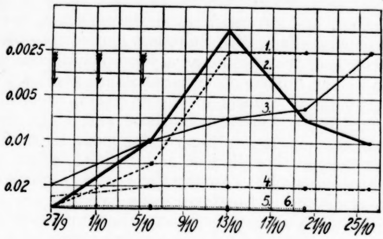
Complement fixation is of no importance in making a diagnosis at the beginning of the illness. However, later on it is important in determining whether a cough, which is not clinically characteristic, is whooping cough or not. The reaction is very valuable at this phase of the illness because it is especially pronounced at that time when most of the bacilli have already disappeared from the expectoration. When a child which has coughed for a long time in a very characteristic way is admitted to a hospital, it may be of interest to ascertain whether or not it has whooping cough, both for the sake of other children, and also to know if it should be placed with other whooping cough patients.

In agreement with the fact that complement fixation appears in whooping cough patients, we note that it is possible to cause an increase in the amount of complement fixation substance in persons vaccinated with whooping cough vaccine.

Here is a curve (6) drawn to represent results of the experiments made by Martin Kristensen. The two uppermost lines show the course of complement fixation after the usual 3 doses of whooping cough vaccine have been injected in the patient. We see that a maximum is reached 8 days after the last injection, after which the line of the curve again falls. This curve resembles far more than the one I just showed you for the patients, the antibody curve which appears after a series of antigens such as diphtheria, typhoid bacilli and others.

CURVE 6

COMPLEMENT FIXATION REACTION
after
WHOOPING COUGH VACCINATION.



(The arrows indicate the days of Vaccine Injections).

No. 3 in Curve 6 is a patient who already had whooping cough when vaccination was commenced. In that case only a small increase in the quantity of antibody was observed in response to the injection of whooping cough vaccine. The curve of the complement fixation substance first rises later, due presumably to the patient's whooping cough. For control, 3 curves are given below from non-vaccinated children, all of whom caught whooping cough later on.

Lastly we reach the question of the *specific treatment* and *prevention* of whooping cough.

Bordet and Gengou have produced a *whooping cough serum* in the usual way by immunizing animals with bacilli. They discovered, however, that this serum had only a slight countereffect on the strongly irritant whooping cough toxin and the results obtained from vaccinating patients with this serum are far from satisfactory. Even though a stray doctor or two reports good results, the majority are of a different opinion, and that particular part of serum-therapeutics has no very extensive field.

Very different results are obtained from *vaccination* with dead whooping cough bacilli. Are the effects prophylactic or therapeutic?

As early as the year 1912 Nicolle and Connor experimented with vaccination in a whooping cough epidemic in Tunis. Of 104 children whose cases could be observed narrowly, 37 were cured, 40 showed improvement, while 27 were unaffected by the treatment. Of the 37 cures, 29 were complete after 2-5 injections, i. e., from 3-12 days. Many doctors, both English and American, but especially the latter, (Freeman, Graham, Saunders, Ladd, Luttinger, Bamberger, Sill, and others) have also used the vaccine therapeutically, and these generally claim that the treatment, when used at an early stage of the disease, and given in rather large doses, has a beneficial effect. Sill, Saunders and many others have used the vaccine as a prophylactic remedy and claim to have observed excellent results. The majority of reports in recent years, especially from North America, England and Italy, confirm the impression that vaccine made from Bordet-Gengou bacilli has a beneficial effect on whooping cough. Among those who recommend it especially is Luttinger whose results are derived from a very large experimental material. He praises the remedy in glowing terms both as prophylacticum and therapeuticum. Appel and Bloom describe in detail from personal experience a whooping cough epidemic in which good results were obtained by using a vaccine compounded of whooping cough bacilli, staphylocci, streptococci, the Pf. bacilli and pneumococci.

Rowland G. Freeman, who was at one time very skeptical in regard to the results of vaccine treatment, wrote in 1920 that fresh vaccine used at an early stage has a prophylactic effect. Davies claims that vaccine mitigates the disease. Miller writes that children are only rendered immune when given large doses. Revalt reports on 130 cases treated with vaccine with splendid results. Paterson and Smellie, on the other hand, saw no good results in 46 cases treated with vaccine.

Herrman and Bell report on a careful study of 300 cases of whooping cough. They write: "The most promising specific treatment is vaccination with the Bordet-Gengou bacilli. In 25% of our cases the improvement was so pronounced that there was no doubt as to the specific effect."

There is no time to discuss in further detail the huge amount of literature already existing on this subject. Yet, on the other hand, as there are still many doctors and hygienists who are skeptical in regard to vaccination, particularly because the results are not supported by suitable control-material, I have felt that the experience we have gained in Denmark might be of interest, based as it is for the most part on material that has been closely watched.

Since 1916 the State Serum Institute has been able to deliver a vaccine made in the following way:

The bacilli of a 48 hour blood-agar culture of Bordet-Gengou bacilli are rubbed off with a fused Pasteur pipette; they are then emulsionized in a 0.5% Phenol-salt water.

Counting shows that the emulsion contains about 10,000,000 Bordet-Gengou bacilli per cm^3.

The vaccine is used as follows: Inject intramuscularly or subcutaneously 3 times with 4 days interval,—the first time use 0.5 cm^3, the second time 0.7 cm^3 and the third time 1 cm^3. In the case of very young patients the doses should be slightly reduced.

Subcutaneous injection often causes local reaction in the guise of infiltration at the point of puncture; in many cases a slight temperature is observed the first 24 hours after vaccination, in a few cases high fever is reported, but no serious symptoms have been observd. Intramuscular injection seems to reduce or exclude the possibility of the above-named inconveniences.

Vaccination has been tried both as a preventive and a therapeutic remedy for whooping cough.

The individuals vaccinated were derived from children's hospitals and doctor's practices, but the largest and most valuable material is from the Faroe Islands. The total number of cases is 2700.

The results may be summarized as follows: No absolutely sure prophylactic effect has been obtained, even though it cannot be denied that in many instances fewer cases of infection are observed among those vaccinated than among the non-vaccinated. There is no doubt that infection is lighter among the vaccinated than among the non-vaccinated.

Let us first consider results of a whooping cough epidemic at a coast hospital in Denmark. Of 62 children who had not formerly had whooping cough, 50 were vaccinated while 12 were left unvaccinated; three of the latter caught whooping cough, a serious case in one instance. Among the vaccinated children 8 cases of whooping cough were found (proved bacteriologically). The disease appeared before the close of the vaccination; the cases were very light, much lighter than the non-vaccinated cases. The epidemic ended here.

At the large epidemic hospital in Copenhagen, Blegdams Hospital, when whooping cough appeared in diphtheria, scarlet fever and other wards, the question arose as to whether the patients should be isolated or not. It was decided to vaccinate the other patients and let the whooping cough patients remain. New patients were also admitted without regard to the danger of contagion, but they were vaccinated at once. The vaccine was given intramuscularly, and at far shorter intervals than is usual, daily for 5 days in succession, in doses of 0.1;0.2;0.4; 0.8; and 1cm^3 of the vaccine. The result was that, of the 50 patients vaccinated, only 6 caught whooping cough, and 2 of these had very light cases. A month later one of the patients vaccinated caught whooping cough.

By far the largest and most important part of the investigations was made under a whooping cough epidemic on the *Faroe Islands*, that tiny little group of islands belonging to Denmark and located in the Atlantic Ocean between the Shetland Islands and Iceland. On account of their isolation they are peculiarly well-adapted to the making of epidemiological observations. Seventeen only of the islands are inhabited; they lie at some distance from each other, separated by straits which often present great difficulties of navigation so that the inhabitants are shut off from each other for months at a time. However, they all know each other intimately, and good medical aid is available. If a contagious disease appears, the source of contagion can almost always be traced, and questions in regard to spread of contagion, time of incubation and conditions of immunity can be readily explained. The Danish doctors of the Faroe Islands have been able to make valuable contributions to the epidemiology of various infectious diseases. Let me recall to your mind that the well-known Danish physiologist, Panum, won his scientific spurs in the valuable information he was able to give about the contagion of measles, based on observations he had made during a large measles epidemic on the Faroe Islands in 1846, which appeared after no measles had been seen on the islands for 65 years.

Whooping cough appears on the Faroe Islands in pronounced epidemics separated by periods of 5-6 years entirely free from whooping cough. It last appeared in 1914-15 culminating in 1915 with 1286 cases. The epidemic was very severe with many complications and 85 deaths. In 1916 it was reported that of 53 cases of whooping cough recorded, 15 died.

It is worthy of note that many of the substantiated cases of whooping cough were second attacks so called "grandmothers whooping cough"; however, these were always light and shorter in duration than the first attacks.

Since 1916 no new case of whooping cough had been observed until a new and severe epidemic began in 1923. At that time the population according to census was 22,000. The doctors agreed immediately to vaccinate on a large scale. On some of the islands the inhabitants were vaccinated as the contagion broke out, on others before it reached them. Two thousand vaccinations were made in all. The Faroe doctors are extremely well pleased with the results of vaccination. The general impression is that while the prophylactic effect is uncertain, there is no doubt but that the attack is much milder than when vaccination is not used. I cannot go through the material in detail, but I will cite a few examples:

A doctor from *Vang* reports that 364 children were vaccinated 1-3 months before being exposed to contagion, in spite of this they all without exception caught whooping cough. However, the epidemic was on the whole very light. In several cases the attack did not advance beyond the catarrhal stage, while those in which the spasmodic cough appeared suffered but little. Three of the 364 patients developed bronchial pneumonia, and a 1½ year old scrofulous child died. Among 30 unvaccinated children, 5 cases of bronchial pneumonia were observed with 1 death.

The district doctor from *Eide* claims that the effect is best seen in children who begin to cough at the earliest one week after the last vaccination day. Whooping cough vaccine caused a decided reduction in the number of complications and in their severity and thus reduced the mortality. Of 108 non-vaccinated, 4 died. Of 251 vaccinated, 1 died. Vaccination checks the power of the disease and shortens its duration. He believes the vaccine to have no effect once the disease has broken out. In regard to the *prophylactic* effect he found that 20% of those vaccinated escaped whooping cough, while of 108 non-vaccinated only 2 failed to catch the disease.

In *Trangisvaag* where 450 were treated, the cough was abortive in several instances and none of those vaccinated died, whereas among 32 non-vaccinated there were 2 deaths.

A doctor in *Klaksvig* reports no deaths among the 457 vaccinated patients. Whooping cough was mild, especially in the case of those patients showing a strong local reaction from the vaccine. There was an equally large control material from Klaksvig, namely 450 non-vaccinated patients, among whom were 11 deaths.

In the total material from this epidemic we note 2094 vaccinated patients with 5 deaths, and 627 non-vaccinated with 18 deaths. The mortality among the non-vaccinated is 12 times as great as among the vaccinated, and the attacks are much more severe. Vaccination is, as a rule, most effective when given about a week before the disease breaks out. Some students of the question claim that the effect is greatest in patients showing a strong reaction at the point of injection. There may be some connection between this fact and the curve showing the formation of antibody in those vaccinated which I have just shown you, and from which it appears that the culmination is reached just a week after the last injection of the vaccine.

Another question, and one which I will not discuss here, is the extent to which this effect of the whooping cough vaccine really is specific, —or whether it is merely due to a protein effect of the injected bacilli.

Summarizing briefly what I have touched upon this evening we see:

(1) Whooping cough is in the lead among

those children's diseases which it is our duty to combat. Statistics prove this only too clearly.

(2) One of the weapons at our disposal is the *bacteriological diagnosis,* which should be established and used in the same way as the bacteriological diagnosis of diphtheria and other infectious diseases is used. I do not know to what extent this is the case in the United States, but in Europe I believe that the diagnostic station in Copenhagen is the only one where physicians employ these methods regularly and on a large scale. An understanding use of this diagnosis would lead to an early isolation of the infected individuals or to the removal of particularly exposed children, especially infants, so long as the danger of infection continues,—that is to say about four weeks after the spasmodic cough has commenced. Complete isolation of all whooping cough patients is, of course, out of the question; we must content ourselves with protecting those who are especially endangered and first of all children under 1 year of age.

(3) Our experience in *vaccinating* for whooping cough challenges us to use that method of treatment to the widest possible extent as soon as a whooping cough epidemic threatens. The health authorities must be on the watch for the first signs of its coming. Everything seems to indicate that we are able to change the character of the infection to a much milder form. It seems advisable to vaccinate as early as possible even after the disease has broken out; in such cases, just as when quick action is necessary, the vaccine should be given in greatly increasing doses five days in succession as described above.

Shall we do away with whooping cough entirely? Of course that is our future ideal, as it is for other infectious diseases whose evil effects are gradually diminishing thanks to the many remedies science now places at our disposal. However, as long as there is no chance of our banishing these diseases from every country of the world, we must be careful not to banish them from a single country, for, by so doing, we run the risk of experiencing what we have observed under the measles epidemic on the Faroe Islands,—when a disease finally returns after a long absence, it attacks young and old without discrimination, and readily assumes that pernicious character so often observed in diseases among a "virgin population."

For the present, at least, it is far better to retain the epidemics under the form of "children's diseases" whose attacks render the entire population practically immune,—and, as we have seen in the case of whooping cough, we have a remedy which takes the poison out of its sting.

SMALLPOX IN THE UNITED STATES, INSULAR POSSESSIONS, NEW ENGLAND AND MASSACHUSETTS, 1913-1923

BY SAMUEL B. WOODWARD, M.D., WORCESTER, MASS.

William Lloyd Garrison, Jr., argued before a Legislative Committee last year that because smallpox was so rare a disease in Massachusetts (300 cases in 10 years) further vaccination of its inhabitants was unnecessary.

Medical Liberty Leagues, incorporated and unincorporated, claim that the disease is receding everywhere, that it is uninfluenced by vaccination, that vaccination itself places the vaccinated one in deadly peril, that smallpox is a filth disease and can be and is cured by pure air and clean living, that we are in a state of "gibbering fear" about it and that "conformation to the spiritualistic in contrast to the materialistic view of life" will protect us from its ravages.

Accepting none of these statements, and convinced that smallpox is more prevalent in the United States at the present time than even physicians, particularly those of New England, appreciate, I have made it my business by personal letters to health authorities of the various states to ascertain the number of cases of smallpox reported to them for the eleven years, 1913-1923 inclusive, and have tabulated the results, arranging the states in the first table according to the member of inhabitants, in the second according to the number of cases of smallpox recorded. To avoid any possibility of error there has been admitted into these tables no case of smallpox unvouched for by the Commissioner of Health of a state or dependency; no *estimates* have been accepted; no state is credited with any cases before the establishment of reports claimed to be accurate by the authorities making them.

In 1913 but 36 states and dependencies kept records. In 1917 48 had fallen into line, and by 1921 a report could be, and was obtained from every state in the Union, Missouri, the last state to tabulate smallpox, making a report in that year and she had 5,400 cases to report. New Mexico, Idaho, Kentucky and Texas began their records in 1920, Wyoming, West Virginia and Oklahoma in 1919, Delaware, Florida, Nebraska, North Carolina and Georgia in 1918, Illinois in 1917, Tennesssee in 1916, Arkansas in 1915, Louisiana and Mississippi in 1914.

Is it, or is it not disgraceful that 130 years after Jenner's immortal discovery there should be reported to the health authorities in these

United States from 21,000 to 109,000 cases of smallpox every year? Is it, or is it not possible to impress upon those who make our laws and those whose duty it is to enforce them when made, that while smallpox is being controlled in other countries, in the United States it is neglected, that it is endemic in many sections and is yearly becoming more virulent? Can nothing be done by the medical profession to bring about a realization of the fact that smallpox *can* be controlled, that the number of cases *can* be reduced to a minimum, that a condition *can* be brought about where for a term of years at least no case will occur unless imported, that the loss of time, of money, of life in certain states of the Union is unnecessary and that if we go on as we are going on smallpox will before long be endemic throughout a large part of the country? It is now and has been for years endemic over a not inconsiderable area.

Is there any reason why in the eleven years 1913-1923 Ohio should have 51,913 cases of smallpox

Washington	24,183
Wisconsin	29,109
Indiana	40,082
Iowa	29,549
Kansas	29,831
Utah	17,852
Oregon	11,999
California	19,702
while Massachusetts had	457
New Hampshire	241
New Jersey	936
Rhode Island	20

Why did Porto Rico with 1,299,809 inhabitants have but 137 cases during this period, while the state of Washington with about the same population (1,356,621) had 24,183? Is it easier to control smallpox in Porto Rico, where it was endemic under Spanish rule, than in the state of Washington? Why did Porto Rico have but 14 cases in the last 6 years, while Georgia had 7,629? Georgia has twice the population and 544 times as many cases of smallpox.

The Canal Zone in 11 years reports but 407 cases, Alaska 5, Hawaii 14, Guam not a case. Compare Rhode Island with Nevada. Rhode Island, small as it is, has nine times the population of Nevada, but had but 20 cases of smallpox to Nevada's 1,142. · Eleven hundred and forty-two cases of smallpox in a population of 77,407! This same proportion of cases in Massachusetts would have given us 57,100 cases. We had 457 The City of New York, including the Bronx and Queens with 3,485,161 inhabitants, had 187 cases in the eleven years. Brooklyn with 2,018,356 inhabitants, 37, or 224 cases for 5,504,617 persons in greater New York, while Ohio with about the same number of inhabitants (5,759,354 to be exact) accumulated 51,913; and this record of New York and Brooklyn, despite the tremendous ebb and flow of outsiders into and out of the former city, is three times better than that of Massachusetts, good as that is in comparison with almost every other state in the Union. Indiana with 2,930,390 inhabitants had 40,082 cases, while New Jersey with 3,155,900 had but 939. Why? And why, again, should Nevada with 77,407 inhabitants have nine times as much smallpox as Porto Rico with 1,299,809, and why should Massachusetts have 457 cases and Michigan with less population 26,972, or Rhode Island 20, Montana 9,861, Utah 17,852, Montana having 50,000 and Utah 150,000 less inhabitants than little Rhode Island? Why once more do New York and Pennsylvania have but 8,134 cases between them, while Kansas, Illinois, Minnesota, Indiana and Ohio, whose combined population is almost exactly equal to the 19,000,000 of New York and Pennsylvania, provide 190,518, although Illinois, having no records back of 1917, is incompletely recorded, and still again why in New England do we find in New Hampshire 241 cases, in Vermont with less inhabitants 1,202? We find Connecticut, with but a little more than a third of the population of Massachusetts and but twice that of its little neighbor, Rhode Island, has 4,493 cases to its credit against the 457 of Massachusetts and the 20 of Rhode Island? Why is this, and lastly one may ask why is it that there is no state in the Northwest, in the Middle West, on the Pacific Slope, and but few in the South that do not report their cases by the thousand, while New England, the Middle States and Maryland are comparatively free from the scourge? Is it possible (*perish the thought*) that the enforcement of vaccination and particularly the vaccination of school children has anything to do with it?

When at a hearing I was told that but four states made successful vaccination a prerequisite for admittance to school and that Massachusetts was one of them, I retorted that Massachusetts had proportionately less smallpox than any state in the Union. The tables show I was not far from the truth when I made that statement. The tables also show that Massachusetts, Rhode Island and New Hampshire, which stand with Hawaii, Porto Rico and Guam in requiring the vaccination of public school children, are in a class by themselves. That Vermont, which has no compulsory law, and Connecticut which entrusts its enforcement to local school committees fail to obtain equally good results, though otherwise subject to the same conditions as the other New England States; that when we come to New York we find 7,199,899 persons living in cities of 50,000 and over with but 668 cases of smallpox credited to them, while the 3,000,000 outside of these cities were credited with 3,801, compulsory pre-school vaccination applying to the former class only, under the New York law.

Utah seems to be the only state with a law specifically forbidding compulsory vaccination as a prerequisite to school attendance. Utah is not a very big state, having but 449,396 inhabitants, but there are but 12 states in the Union that reported more smallpox and not one of these but had at least three times as many inhabitants.

My object in tabulating these figures is not to prove a thesis but to arouse interest in the problem of smallpox to show that it is a problem and that 579,610 cases of the disease in the imperfect reports of eleven years are not to be sneered at even by the 107,460,178 persons involved.

But were I desirous of showing the importance of vaccination and particularly the vaccination of school children I would call attention to the fact that the Sanitary Code of the Canal Zone (Panama and Colon included) calls for the vaccination of all inhabitants, that since 1913 there have been five years with no cases of smallpox and that an epidemic starting in 1918 and stamped out after a four years' fight accounts for all but 10 of the 407 cases reported, and this despite the fact that smallpox is endemic in all contiguous territory. If I were writing for anti-vaccinationists instead of to physicians, I would state that every child in Porto Rico must be vaccinated before it is a year old, that every child whether successfully vaccinated or not must be revaccinated at the age of seven, that if unsuccessful this must be repeated in a month, and if still not successful, the child must be revaccinated between its ninth and tenth years, and that it is further provided that those persons not twenty years of age who have been successfully vaccinated shall be again vaccinated eight years after the last vaccination, and, if not successful, again at the end of a month, that all teachers, employees and care-takers of both public and private schools, all employees of railroads, tramways and public vehicles, all proprietors and clerks of hotels and restaurants, all managers and operatives in workshops and factories must produce certificates according to their ages guaranteeing that they have been vaccinated or revaccinated, as the case may be, and that no child can attend school without such certificate.

And were I writing to impress legislators, I would say that no state in the Union, with the exception of Rhode Island, has during the last eleven years been so free from smallpox as has Porto Rico, that the states with approximately equal populations, namely, Nebraska, Washington, Connecticut, Maryland and West Virginia, report while Porto Rico had but 137—109 of these in one epidemic lasting but a few months in 1916. I would say that Porto Rico stands head and shoulders in its immunity above every place from which I have records, bar Guam, Alaska, Hawaii and Rhode Island, and this in an island where under Spanish rule smallpox was not only a constant visitor but a permanent resident.

Nebraska (since 1918)	14,521	cases
Washington	24,183	"
Connecticut	4,493	"
Maryland	1,867	"
West Virginia (since 1919)	9,321	"

Porto Rico was never free from smallpox until it was vaccinated by the Army of Occupation in 1898.

I might say also in passing that Hawaii had seven cases of varioloid in 1913 and that for the last ten years no case of smallpox has appeared in the Sandwich Islands, bar seven which were removed from visiting ships at quarantine; that Hawaii vaccinates its school children; that in Guam, isolated as it is, they are taking no chances, having compulsory school attendance at the age of seven, and admitting no unvaccinated child to the school room. (No case of smallpox there for at least eleven years.)

Guam, Alaska, Hawaii, Rhode Island, Porto Rico, New Hampshire, Delaware and the Canal Zone are the only divisions of the country that individually report less smallpox than do we, and as the combined populations of the sections cited is nearly a million less than that of Massachusetts, while the smallpox incidence is more than twice that of our home state, we can hold up our heads unembarrassed and unalarmed, even though we know that had we no more cases than Rhode Island in proportion to our respective populations, we should reduce our morbidity from 457 to 120. Had we the same laws as the City of New York, perhaps we could do this, but that is another story.

Health authorities, practically without exception, have cheerfully given me what I have asked of them.

The statistics of the Philippine Islands were not available when the foregoing was written but have come to hand and appear below so that with the exception of the Island of Samoa I present a complete record of smallpox in every part of the United States for the last three years and a more complete statement than has ever been made public, so far as I know, for the last eleven. I think it worthy of study. I think it encourages us to keep up the fight, to keep up the bars in Massachusetts, and hold the state where she is in the forefront of those striving to protect the country from what seems to me almost inevitable, and that is, a terrific eventual epidemic of smallpox, for the West will not learn, the anti-vaccinationists are active, and the people are asleep at the switch.

APPENDIX

Manila, November 1, 1924.

Dear Sir:

Your letter received and I have referred it to the Philippine Health Service to send you the necessary

SMALLPOX FOR ELEVEN YEARS CLASSIFIED ACCORDING TO POPULATION

State or Territory	Population	1913	1914	1915	1916	1917	1918	1919	1920	1921	1922	1923	Total
Guam	16,225	0	0	0	0	0	0	0	0	0	0	0	0
Alaska	54,899												5
Nevada	77,407	256	41	81	6	21	22	89	321	235	59	11	1,142
Canal Zone	122,713	0	0	0	2	0	126	63	24	184	8	0	407
Wyoming	194,402							414	911	486	179	20	2,060
Delaware	223,003						40	259	27	0	0	2	328
Hawaii	255,912	7	0	0	0	0	0	0	4	0	2	1	14
Arizona	334,162	203		91	49		200	69	176	192	468	100	1,548
Vermont	352,428	138	25	214	152	66	55	5	101	177	14	255	1,202
New Mexico	360,350								311	110	53	91	565
Idaho	431,866								2,556	894	241	155	3,846
Dist. Columbia	437,571	140	150	15	4	11	48	173	89	66	66	67	829
New Hampshire	443,083	113	10	16	1	2	27	14	0	19	0	39	241
Utah	449,396	2,443	1,139	456	46	900	2,263	1,238	5,123	3,499	578	167	17,852
Montana	548,889	725	701	546	569	1,346	1,104	970	1,066	1,466	636	732	9,861
Rhode Island	604,397	0	0	2	0	0	2	5	1	1	9	0	20
South Dakota	636,547	658	425	961	93	795	920	1,050	1,534	1,959	1,141	249	9,785
North Dakota	646,872	395	570	708	472	529	345	211	533	1,777	657	444	6,641
Maine	768,014	255	394	4	0	621	569	439	246	96	36	118	2,778
Oregon	783,389	1,024	458	585	120	122	499	2,626	2,991	1,596	1,141	837	11,999
Colorado	939,629	268	440	200	103	323	1,680	1,715	2,898	2,606	1,141	90	11,464
Florida	968,470						59	45	428	1,351	318	227	2,428
Nebraska	1,296,372						3,906	2,844	4,135	3,055	484	97	14,521
Porto Rico	1,299,809	4	1	8	109	3	0	1	2	1	8	0	137
Washington	1,356,621	1,620	785	254	637	1,016	1,676	4,390	6,146	4,441	1,595	1,623	24,183
Connecticut	1,380,631	190	1,969	22	175	1,528	94	6	2	8	447	52	4,493
Maryland	1,449,661	182	541	94	69	98	219	229	179	214	17	25	1,867
West Virginia	1,463,701						1,266	2,214	2,592	2,443	453	253	9,221
South Carolina	1,683,724	683	207	394	97	188	214	304	1,118	1,084	342	544	5,175
Arkansas	1,752,204			482	1,066	2,016	2,253	588	842	485	158	315	8,205
Kansas	1,769,257	812	854	2,861	2,085	3,490	7,130	2,437	3,900	4,927	854	481	29,831
Mississippi	1,790,618		1,173	2,468	1,402	1,530	3,601	2,502	3,148	2,161	729	292	19,006
Louisiana	1,798,509		585	413	819	834	950	1,226	1,564	1,421	645	693	9,150
Oklahoma	2,028,283							2,090	2,422	1,365	736	1,164	7,777
Virginia	2,309,187	1,771	3,538	1,143	462	636	1,783	2,353	3,559	1,737	513	409	17,904
Tennessee	2,337,885				178	1,108	3,217	1,874	3,002	2,913	567	1,037	13,896
Alabama	2,348,174	926	69	775	157	821	3,647	935	1,442	2,072	623	323	11,790
Minnesota	2,387,125	2,853	1,847	1,708	1,256	2,827	2,285	2,448	6,333	9,375	2,270	1,950	35,152
Iowa	2,404,021	2,138	3,260	2,626	1,382	2,133	3,292	1,784	4,485	6,677	1,104	668	29,549
Kentucky	2,416,630								1,792	2,367	470	228	4,857
North Carolina	2,559,123						983	2,322	2,961	2,513	1,409	3,352	13,540
Wisconsin	2,632,067	2,034	3,302	1,817	867	1,280	2,600	3,297	5,668	5,116	1,790	1,338	29,109
Georgia	2,895,832						971	1,710	1,328	2,191	756	673	7,629
Indiana	2,930,390	3,110	4,522	3,456	1,158	4,832	5,582	3,833	5,775	4,809	1,095	1,910	40,082
New Jersey	3,155,900	86	23	156	9	6	65	109	182	255	18	30	939
Missouri	3,404,055									5,400	675	316	6,391
California	3,426,861	800	677	336	248	329	1,100	2,002	4,497	5,557	2,127	2,029	19,702
Michigan	3,668,412	1,995	1,527	1,094	1,581	3,674	5,241	2,381	4,818	1,120	1,230	2,311	26,972
Massachusetts	3,852,356	157	38	24	32	65	27	40	29	37	2	6	457
Texas	4,663,228								2,113	1,781	1,427	669	5,990
Ohio	5,759,394	2,633	4,789	3,221	2,184	5,258	10,389	4,107	7,209	7,286	2,422	2,415	51,913
Illinois	6,485,280				3,385	4,709	3,842	4,871	8,536	8,536	2,116	930	36,925
Pennsylvania	8,720,017	798	748	117	97	370	611	229	215	212	85	183	3,665
New York	10,385,227	823	791	155	30	362	534	191	305	631	255	392	4,469
Total	107,460,178	30,240	35,599	27,503	21,102	43,849	75,437	62,602	109,639	108,904	34,169	30,313	579,610
Number		36	37	39	40	40	47	49	53	54	54	54	

reports concerning smallpox in the Philippines, which I hope will reach you safely.

Very respectfully,
LEONARD WOOD,
Governor-General.

Mr. Samuel B. Woodward,
President, Worcester County Institution for Savings, Worcester, Massachusetts.

OFFICE OF THE GOVERNOR-GENERAL
OF THE PHILIPPINE ISLANDS

Manila, November 13, 1924.

Sir:

Referring to the Governor-General's letter to you of November 1, 1924, acknowledging receipt of yours of September 30, 1924, requesting smallpox statistics for 1913-1924, I have the honor to quote the following indorsement of the Director of Health of November 8, 1924:

> "Respectfully returned to His Excellency, the Governor-General of the Philippine Islands, thru the Honorable, Secretary of Public Instruction with the requested data herewith furnished.
>
> "Prior to the year 1918, cases of smallpox in the provinces were not regularly reported and recorded. For this reason, exact data on cases prior to the year 1918 are not available in our record. Deaths, however, are on hand, which we here furnish. At the close of the

year this Office will furnish Mr. Samuel B. Woodward with the total smallpox cases and deaths for 1924."

The data referred to above is attached.

Very respectfully,
J. M. Woolfolk,
Assistant Secretary to the Governor-General.

Mr. Samuel B. Woodward,
President, Worcester County Institution for Savings, Worcester, Massachusetts.

Philippine Health Service
Division of Communicable Diseases
Manila

Cases and Deaths from Smallpox Registered in the Philippines During 1913-1923, Inclusive

Years	Cases	Deaths
1913	(?)	903
1914	(?)	438
1915	(?)	273
1916	(?)	610
1917	(?)	436
1918	47,369	16,567
1919	65,180	49,971
1920	15,857	7,105
1921	1,858	726
1922	128	19
1923	69	3

Note—(?) means cases were not regularly reported and hence not recorded (no data).

Smallpox Classified According to Cases for Eleven Years—1913 to 1923, Inclusive

State	Population	Cases	Less Than Eleven Years
Guam	16,225	0	
Alaska	54,899	5	
Hawaii	255,912	14	
Rhode Island	604,397	20	
Porto Rico	1,299,809	137	
New Hampshire	443,083	241	
Delaware	223,003	328	6 years
Canal Zone	122,713	407	
Massachusetts	3,852,356	457	
New Mexico	360,350	565	4 years
District of Columbia	437,571	829	
New Jersey	3,155,900	939	
Nevada	77,407	1,142	
Vermont	352,428	1,202	
Arizona	334,162	1,548	8 years
Maryland	1,449,661	1,867	
Wyoming	194,402	2,010	5 years
Florida	968,470	2,428	6 years
Maine	768,014	2,778	
Pennsylvania	8,720,017	3,665	
Idaho	431,866	3,846	4 years
New York	10,385,227	4,469	
Connecticut	1,380,631	4,493	
Kentucky	2,416,630	4,857	4 years
South Carolina	1,683,724	5,175	
Texas	4,663,228	5,990	4 years
Missouri	3,404,055	6,391	3 years
North Dakota	646,872	6,641	
Georgia	2,895,832	7,629	6 years
Oklahoma	2,028,283	7,777	5 years
Arkansas	1,752,204	8,205	8 years
Louisiana	1,798,509	9,150	10 years
West Virginia	1,463,701	9,221	5 years
South Dakota	636,547	9,785	
Montana	548,889	9,861	
Colorado	939,629	11,464	
Alabama	2,348,174	11,790	
Oregon	783,389	11,999	
North Carolina	2,559,123	13,540	6 years
Tennessee	2,337,885	13,896	8 years
Nebraska	1,296,372	14,521	6 years
Utah	449,396	17,852	
Virginia	2,309,187	17,904	
Mississippi	1,790,618	19,006	10 years
California	3,426,861	19,702	
Washington	1,356,621	24,183	
Michigan	3,668,412	26,972	
Wisconsin	2,632,067	29,109	
Iowa	2,404,021	29,549	
Kansas	1,769,257	29,831	
Minnesota	2,387,125	35,152	
Illinois	6,485,280	36,925	8 years
Indiana	2,930,390	40,082	
Ohio	5,759,394	51,913	

BLINDNESS FROM TEETH, TONSILS AND ACCESSORY SINUSES*

BY LEON E. WHITE, M. D.

The word "blindness" at once suggests a deprivation so overwhelming that many would choose oblivion rather than existence without that wonderful gateway through which pass most of the impressions making life worth while, and without which the struggle for existence becomes so handicapped that not a few are compelled to seek charity to keep body and soul together. The hope that some of these victims would be benefited by a better understanding of the whys and wherefores of loss of vision from infections in the sinuses, teeth or tonsils has stimulated me to continue the investigation commenced twelve years ago. To this end various phases of the subject have been studied and papers read and published on the anatomical relations of the optic nerve, the literature, the diagnosis, the prognosis, the etiology, the pathology, treatment by aëration, etc., and more recently two papers one "An Anatomical and X-ray Study of the Optic Canal," and the other "The Treatment of Optic-nerve Involvements as Determined by Optic Canal Radiographs." Within these various papers are detailed reports of 59 cases.

Since learning how to measure the optic canals, some of the views expressed in earlier papers have been modified and are here reconsidered. But first let me give you a few extracts from the literature, which is voluminous and dates back to Beer in 1817. In 1886 Berger and Tyrman gave a brief review of the previously reported cases of blindness, some 26 in number, arising from sphenoidal disease, and noted that the bony wall separating the nerve from the sinus was frequently of only tissue paper thickness.

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*Read before the Maine Medical Society, Eye, Ear, Nose and Throat Section, at Portland, Me., on February 7, 1924.

Onodi, in 1908, said: "Our observations have shown that the wall between the last ethmoid cell and the canalis opticus is nearly always as thin as tissue paper; dehiscences in the walls of the accessory cavities have been found; there the diseased mucosa may come into direct contact either with the dura mater or the optic-nerve sheath."

Normally, according to Loeb, "the optic nerve may be described as passing externally from the chiasm along the roof or lateral wall of the sphenoid and in close relation with the ethmoid labyrinth *only* at the posterior external angle of the last cell."

From Schaeffer's book on *The Nose and Olfactory Organ* the following is quoted: "It is essential that the intimate anatomic relationships which exist between the paranasal sinuses and the optic nerve and commissure be understood by ophthalmologists and rhinologists. It is established that disease of the paranasal sinuses may lead to an optic neuritis, even to blindness. Of the paranasal sinuses the sphenoidal and the posterior ethmoidal especially concern us in this connection.

"After the optic nerve passes beyond the vicinity of the posterior ethmoidal cell it diverges more and more from the ethmoidal field and is no longer in intimate relationship with the other ethmoidal cells." This brief review suffices to show how thoroughly the relations of the optic nerve to the accessory sinuses has been worked out so that no one can question the fact that the sphenoids and posterior ethmoids are the only sinuses in intimate relation with the optic nerve, and to reach the tissue adjacent to the nerve the direct and logical route would be through these structures and not through the entire ethmoidal labyrinth.

The diagnosis is naturally of major import. The symptoms in the ordinary cases of acute optic or retrobulbar neuritis are quite typical. Associated with the loss of vision there may be discomfort about the eye or lameness on moving it. Occasionally there is slight exophthalmos, this condition indicating an inflammatory process in the orbit. Pupillary changes and ptosis are found occasionally. There is frequently nyctalopia, a central scotoma for colors, enlargement of the blind spot and contraction of the fields. The patients generally say that things at first were blurry when looking straight ahead, but comparatively clear when viewed sideways. Changes in the fundus are of great diagnostic value, varying, as they do, from the normal to a commencing pallor in some cases, and in others to a marked redness and swelling about the nerve, with engorgement and tortuosity of the retinal veins. It is essential that these inflammatory swellings be differentiated from the edema, due to intracranial pressure.

The anterior portion of the nose frequently appears normal. The one vital point to determine, in the nasal examination, is the size and position of the middle and superior turbinates. Do they block the ventilation of the posterior sinuses? Is there impaired aëration? It takes but a slight obstruction to interfere with the ventilation, and the mistake is frequently made of expecting to discover marked changes; while, on the other hand, whenever there is no blocking of the normal openings of the posterior sinuses, this region can, I believe, be ruled out as a causative factor in the neuritis.

The following is quoted from Stark, who expresses well the thought I wish to convey: "From a nasal standpoint we must not expect to find the common symptoms of sinus infection, pus, polypus, history of nasal discharge, etc., as we are dealing with a closed sinus; otherwise we should not have pressure. * * * * The deflected septum and middle turbinate tightly pressed against the lateral wall should always be suspected."

Over half the cases give a history of a recent coryza or a prior influenza, but in a few there is no history of infection. It is impossible to determine dental infection by a superficial examination of the teeth, many appearing normal even when abscessed, so that no examination is complete without a careful filming of every tooth. It is not always possible to determine the health or disease of a tonsil. Any enlarged cervical gland below the angle of the jaw is practically diagnostic. Congestion about the pillars or soft palate usually indicates infection. It is imperative that careful radiographs should be made of the accessory sinuses and optic canals. Whenever the sinuses are negative the teeth and tonsils should be suspected. Tonsils at all suspicious, with teeth and sinuses negative, should be removed. The diagnosis many times must be made largely by exclusion. In all cases the patient should undergo a thorough physical and neurological examination. One should consider in turn blood, urine, hysteria, pellagra, lues, tobacco, alcohol, lead, arsenic, quinine, etc. While investigating, time is a very important element, and it is essential that needless delay be not encountered. Practically all necessary tests can be made within forty-eight hours. Pituitary disease, brain tumor and multiple sclerosis, while not usually producing such *sudden* loss of vision, must always be borne in mind. As a matter of routine the various cranial nerves and lobes of the brain are tested. Not a few cases with brain tumor have had various nasal operations before they were correctly diagnosed. Within the past year I have been guilty of removing tonsils and opening the posterior sinuses in 2 cases, but not until they had been thoroughly investigated at the Peter Bent Brigham Hospital and discharged on the assumption that there was no intracranial condition to account for the loss of vision. These cases, while not typical of accessory sinus blindness, had sufficient pathology in the nose and throat to warrant making an attempt to benefit their vision by this procedure. They were not benefited and both have since been operated upon for suprasella tumors. The knowledge derived from these patients has greatly assisted me in correctly diagnosing 3 recent cases with intracranial tumors. Careful investigation should prevent unnecessary operating. One should systematically go over each case before advising operation and convince himself that the eye condition in all probability originates from that which he proposes to open or remove, be it accessory sinuses, teeth or tonsils.

ETIOLOGY. The earlier writers considered the mere presence of pus in the sinuses the all-sufficient explanation for the disturbance about the optic nerve, and while this may undoubtedly be the cause in some instances, it is by no means the usual or the only one. The nerve is ordinarily protected by the barrier thrown out by inflammatory processes, so that it is rarely involved unless through some anatomical abnormality. In only a small percentage of my cases has pus been found. Hyperplasia has been emphasized by others as the chief cause, and while hyperplastic tissue may involve the posterior sinuses, it has seemed to me, from my study of the sections and cases, that it would better be considered a predisposing factor rather than the principal etiologic conditon. Hyperplasia undoubtedly renders the sinuses more vulnerable. The etiology seemed to be explained, in most of my cases, either by the size and position of the middle and superior turbinates or from infection in the teeth or tonsils.

PATHOLOGY. I have undertaken the consideration of this subject under five heads, namely:

1. Direct extension.
2. Toxemia from some infective process.
3. Bacteremia or focal infection.
4. Hyperplasia.
5. Anaphylaxis.

1. By direct extension infections in the posterior sinuses probably extend by continuity of tissue to the optic nerve. When, however, there is an infection in a more remote sinus (an antrum for instance) it may become walled off before the nerve is reached. Should the infection persist, there may be forced into the system a certain amount of bacteria and toxins, which, in turn, can produce an optic neuritis.

2. Toxemia. It is conceded that retrobulbar and optic neuritis can be caused by alcohol, lead, tobacco, quinine, optochin, arsenic, lues, etc., so that, reasoning by analogy, there is little doubt but that toxins originating in the accessory sinuses, teeth or tonsils, or, for that matter, anywhere in the body, may have similar action on the optic nerve.

3. Bacteriemia. It has been demonstrated by Billings that infectious microörganisms may be carried in the blood stream or by the lymph channels from the foci of infection in the teeth, tonsils and accessory sinuses to the terminal bloodvessels in various regions of the body. He has shown how the inoculated bloodvessels become more or less occluded by endothelial proliferation and leukocytic infiltration, and that the bacteria escape through the vessel walls into adjacent tissue, so there would seem little doubt but that bacteria may also travel via the blood stream and lymph channels to the optic nerve.

4. Hyperplasia. As a predisposing factor, hyperplasia is probably of considerable importance. It undoubtedly renders the accessory sinuses more vulnerable. Hyperplasia plus infection and direct extension to the optic nerve is probably of far greater consequence than the mere fact that the tissue has become hyperplastic.

5. Anaphylaxis. Stark has advanced the theory that there is a "sensitization of the tissues of both the sinus and the orbit by the bacterial proteins, producing an allergy resulting in a localized anaphylactic reaction each time the individual comes in contact with a fresh infection of the same bacteria in the nose, and possibly in other parts of the body. For that reason many of these cases give a history of attacks resembling hay fever or acute coryza shortly previous to the eye trouble." While I have had a few cases presenting symptoms similar to those mentioned by Stark, it had not occurred to me that this would satisfactorily explain the pathology. It is, I believe, a valuable suggestion. There certainly seems to be a similarity between some of these eye conditions and certain anaphylactic reactions comparable to asthma and hay fever. As sinus infections frequently cause asthma, it is conceivable that they might also produce engorgement about the optic nerve.

PROGNOSIS. The prognosis depends largely upon the duration and extent of the loss of vision, the condition of the fundus, the virulence of the infection and the size of the optic canal.

Duration. The conclusions reached from an analysis of the cases were that unless there was improvement under treatment within a week there was danger of permanent impairment of vision, while in cases of more than two months' standing little could be expected except possibly checking the loss of vision by the removal of some definite focus of infection.

The Amount of Vision. It was also found that the demand for early operative interference in total loss of vision was more imperative than when the loss was but partial.

Condition of the Fundus. When the nerve appears normal one might, with safety, delay operating much longer than where there is increasing engorgement or commencing pallor.

The *Virulence of the Infection.* As in all the other types of infection, so in that producing optic neuritis, the microörganisms differ greatly in virulence. When the infection is of the virulent type there is probably considerable exudate about the nerve or even within its sheath. The optic nerve, as you know, is really not a nerve but a part of the brain. It is easily destroyed and does not regenerate. Parsons says of it: "The so-called optic nerve, together with certain parts of the retina, constitutes a lobe of the brain, and has, therefore, the characteristics of the central nervous system. Hence the nerve fibers are devoid of a sheath of Schwann and the interstitial substance is neuroglia."

The *Size of the Optic Canal.* That the osseous optic canals normally afford great protection to their neural contents cannot be doubted, but when these canals become narrowed and the enveloping sheath of the nerve, or the nerve itself, swollen the unyielding walls may cause constriction sufficient to produce atrophy. Before mentioning the treatment, I wish to discuss the size and shape of the optic canal, as it is a new subject and one to which I have devoted many hours within the past year. Piersol gives the diameter of the optic nerve as from 3 to 4 mm. In a canal of 4 mm., therefore, only a moderate swelling would cause a pressure so severe that degeneration of the nerve would rapidly ensue. The importance of filming the optic canal to determine adjacent pathology has been emphasized by van der Hoeve. This study was undertaken partially to substantiate his assertions, but more especially to determine if there was any relationship between the size of the canal and the vulnerability of its contents. About 200 skulls were examined at the Harvard Medical School and the results tabulated; the size, shape and position relative to pneumatization were noted. Roentgenograms were made of the optic canals in all available cases of optic-nerve involvements, old and new, to determine the size and shape in the old cases and in the new ones to also ascertain any evidence of pathology. As a check, radiograms were made of 25 supposedly normal individuals. For this X-ray work I am indebted to Dr. A. S. Macmillan.

Up to the present, with the exception of pituitary cases, I have been able to find in the X-ray plates but little evidence of pathology adjacent to the optic canal. Possibly this is due to the type of infections largely responsible for the neuritis, as these happened to be mostly from teeth and tonsils. I regret, moreover, that I cannot agree with van der Hoeve as to the importance of finding purulent sinusitis adjacent to the canal. It has been rarely possible to discover acute infections within the posterior sinus by the X-ray, while in the chronic cases which show pathology in the film, Nature has walled off the infection from the nerve.

The important thing to determine is the size and shape of the optic canal. Adjacent pathology, while always sought, is not so easy to determine and many cases undoubtedly require surgical interference where it is not evident. The average diameter of the optic canal is given by Whitnall as from to 5 to 6 mm., while Wilbrand and Saenger give the average as high as 7 mm. The canals were measured in 164 adult skulls, the average diameter being 5.17 mm. The 5.5 and 6 mm. canals were all round and only 2 had extensive pneumatization about them. Seven of the 164 canals of 5 mm. were slightly oval.

Four were triangular and but 12 showed extensive pneumatization. Of the 4.5 mm. canals (20 in number) 12 were oval or flattened at the top and practically all were two-thirds to three-quarters surrounded by pneumatic sinuses. The 3 canals of 4 mm. were all oval or irregular. Two were nearly surrounded by pneumatized sinuses. The third was in a very dense skull and was probably due to hyperostosis.

The canals were usually found to be round irrespective of the way examined, *i. e.*, by holding them up to the light, by the fluoroscope or the X-ray. The top posteriorly was occasionally flattened and in a few cases the canal appeared distinctly oval, with the horizontal diameter usually the larger. Early in the measurements it became apparent that excessive pneumatization of the sphenoid adjacent to the canal was usually associated with diminution in its caliber. The thinness of the bone separating the inner wall of the optic canal from the sphenoid was noticeable in many specimens, and in a few dehiscences were found, but as these might have resulted from cleaning the skulls, it was thought best not to tabulate them.

The size of the optic canal in infants and children is most instructive. The skulls of 40 stillborn infants were examined fluoroscopically. Of these it was possible to make out the canal in 20. In 2 the canal was oval and in the rest approximately round. In none was it less than 4½ mm. in diameter.

Thus it would appear that the diameter of the optic canal at birth is nearly of adult size, but very short. There is a rather close analogy between it and the annulus tympanicus. The canal seems to reach adult size early in life, judging by measurements obtained from a few children's skulls. Of 6 canals in skulls from children under three years, 4 were round and 2 slightly oval, and all approximately 5 mm. in diameter. The 4 canals from skulls seven to eight years old were all round, and all of about 5 mm. The canals did not appear in these few specimens to be in very intimate relationship to the pneumatic sinuses. Uniformity in the radiograms was obtained by having all the work done by one radiologist. To obtain the normal size and shape of optic canals, 25 doctors, nurses and attendants at the infirmary were filmed. Five of the 50 canals in this normal series were slightly oval, *i. e.*, the vertical diameter was ½ mm. shorter than the horizontal. The average diameter was 5.35 mm. Twenty-five cases with optic-nerve involvement were then filmed; 26 of these canals were oval. This makes 50 per cent oval as against 10 per cent in the normal series. Several of these were filmed a second or third time in an endeavor to get correct positions. In 10 there was 1 mm. difference between the vertical and horizontal diameters. In the others about ½ mm. In 19 of these 25 the optic nerve was involved. The average diameter of the canals of the patients with optic-nerve involvement was 2/3 mm. less than the normal series.

The following is Dr. Macmillan's description of the correct position for filming the optic canal, The position in which a patient is placed for a radiograph of the optic canal is of extreme importance, as a slight variation in the position would so change the outline of the canal as to render its measurements worthless. Holding a dry skull in the hand, and looking at the optic canal through the orbital cavity, it is evident that the true shape and size can be seen only when the axis of the optic canal goes through the lower outer quadrant of the bony rim of the orbit. The optic canal in the living can be thrown into this area when the patient is lying face down with the malar bone, nose and lower jaw touching the plate, the central ray directed straight down. If the upper rim of the orbit is allowed to touch the plate the optic canal will be thrown too high in the bony frame of the orbit and will be distorted. The diameter of an optic canal of 5 mm. would be enlarged about ½ mm. in the radiograph of a living subject.

In a study to determine what influence the size of the canal had on the best method of treatment, 36 cases with optic-nerve involvement were analyzed.

SUMMARY OF THE FINDINGS IN 36 CASES OF OPTIC-NERVE INVOLVEMENT. In 10 cases the vertical diameter was 4 mm. or less, and all were oval. Two of these were benefited but slightly, if at all, by operation. One unoperated case had complete optic atrophy. Another refused operation and was unimproved. Four cases improved greatly after the opening of the posterior sinuses, but there remained marked temporal pallor. One required a tonsil operation, and the remaining case, unoperated upon because of poor general health, has had frequent recurrences. These were by far the most serious cases in the series.

There were 10 cases with canals approximately 4.5 mm in the vertical diameter, and of these but 3 were oval. Four had the posterior sinuses opened and all recovered with practically normal vision. Five had teeth or tonsils removed, and all these made good recovery. The remaining case recovered under local treatment. It is interesting to note that in these 4.5 mm. cases every one recovered with practically normal vision.

There were 11 cases with optic canals 5 mm. in the vertical diameter, and of these but 2 were slightly oval. In 3 chronic cases, where the etiology was in doubt, 1 showed marked improvement after removing the tonsils. A septum and tonsil operation on the second was followed by slight improvement. The third case, with progressive blindness, was temporarily improved in health and relieved of severe headaches, but later had a brain tumor removed. Four acute cases had the posterior sinuses opened; normal vision in 3 and but slight improvement in the fourth. The neuritis in this case was apparently not of nasal or dental origin. Three acute cases recovered under local treatment. The remaining case made a good recovery following the removal of the tonsils and middle turbinate. Of the 8 acute cases, 7 recovered with normal vision, and in the obscure eighth case tabes or multiple sclerosis is suspected.

There was 1 case with canals 5.5 mm. in diameter. In one eye there was optic atrophy of many years' duration; the other eye, of six weeks' standing, improved rapidly after the removal of abscessed teeth.

There were 3 cases with canals abnormally large, *i. e.*, 6 mm. One of these, a case with progressive loss of vision, continued to fail after the sinuses were opened and the tonsils removed. The neuritis was later found to be due to a suprasella tumor. In the second case the vision returned to normal after the removal of the foci of infection in the mouth. The third case, untreated, had complete optic atrophy of two years' duration, probably from either abscessed teeth or an optic-nerve tumor.

Further analysis of this data reveals that in the 10 cases with canals of 4 mm. or less, diseased teeth were extracted in one and in another the tonsils were removed.

TREATMENT. If future cases substantiate the results in those already studied, it will mean that whenever we are dealing with canals of 4 mm. or less the appropriate treatment in a case of severe optic-nerve involvement will be the immediate ventilation of the posterior sinuses to prevent atrophy, unless some other definite focus can be found.

A 4.5 mm. canal does not demand such radical treatment. Optic atrophy, while possible, is less to be feared. The posterior sinuses, unless blocked or evidently diseased, rarely need to be opened. Local medication may be given a reasonable trial. In canals of 5 mm. or over the neuritis is probably not a direct extension from the sinuses, and recovery from any acute attack may be expected either spontaneously or under local treatment. Later the focus should be sought and, if found, eliminated, be it in teeth, tonsils or sinuses, as the toxins from long-continued infection can produce atrophy even in normal canals. Local treatment is usually a 10 per cent spray of argyrol and adrenalin about the middle turbinate, followed by hot irrigation or suction. When no definite explanation can be found for sudden and persistent loss of vision in a patient with a canal of 4 mm. or less, the operation advocated as a therapeutic measure is the removal of the middle turbinate, a larger opening in the front wall of the sphenoid and the uncapping of the posterior ethmoid cell—a simple and comparatively safe surgical procedure to one at all skilled in intranasal technic. The complete ethmoid exenteration, unless the cells are definitely infected, does not seem to me either a justifiable or necessary procedure.

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MEDICAL ATTENTION IN CONNECTION WITH REHABILITATION OF INJURED EMPLOYEES*

BY JOSEPH A. PARKS, MEMBER, INDUSTRIAL ACCIDENT BOARD OF MASSACHUSETTS

Prior to the awakening to the need of conserving life, there was little organized attention given to the man incapacitated by the warfare of industry. When a man was injured, he collected damages or insurance, oftentimes inadequate in amount, and practically no attention was given to the question of his rehabilitation. He was left to fight his life-battle with his handicap, and to overcome it as best he could. He fell upon the human scrap-heap, a living sponge from which had been squeezed all hopes and ambitions, and frequently all but the mere thread of life itself. The lack of advanced surgical attention in the "first-aid" stages of the injury sometimes left a monument of a neglectful State.

There was no wonder at this apathy in treatment of a serious problem, for a constant stream of labor was flowing into our country from all parts of the world. It was absorbed into our industry as so much raw material, and one more or less thrown upon the community as a worn-out cog from the wheels of industry meant little to our onrushing material development. This stream has ceased to flow towards us.

The passage of Workmen's Compensation Acts in the various states has brought home to employers the realization of their duty to men and women injured in industry.

I have been a member of the Industrial Accident Board of Massachusetts since its inception in 1912 and every day I am brought face to face with the terrible consequences of serious injuries. I cannot help feeling that the most important function of the Industrial Accident Board should be to restore to these victims of industrial accidents their old feeling of self-assurance and renewed interest in living so that they may be restored to economic usefulness.

It is true that the Workmen's Compensation Act comes to the aid of these unfortunates for a time, but after a few years the benefits are exhausted and the injured man is entirely cut off in the prime of manly vigor from the work he knows so well how to do. He sees no occupation open to him and it seems to me that everything surgical skill can do for him should be done so that he may be able to take up some form of remunerative employment.

In my opinion, medical attention plays the most important part in the rehabilitation of an injured employee. Without immediate and constant expert medical attention the injured man is liable to become a cripple for life, and not only costs the insurance carrier but his employer the maximum amount payable under the Compensation Act.

When the Compensation Act was first passed little or no attention was given to the medical phase of it. Most of the states allowed, as we did in Massachusetts, medical attention for the first two weeks. At the end of that time the employee, in most instances, was left on his own resources, and sometimes he went without any attention. In other instances he received it in such a haphazard way that it was of little or of no benefit to him. It took only a few years for insurance companies and employers to realize that in order to reduce the ratio payments of compensation benefits they must do something to care for the injured man medically.

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*Read at the Eleventh Annual Meeting of the International Association of Industrial Accident Boards and Commissions, Halifax, Nova Scotia, August 26-28, 1924.

Insurance companies in Massachusetts have come to realize that the best in medical service is cheapest in the end, as competent and adequate treatment of injuries returns the employee to his work in the shortest time and in the best possible condition, thus reducing to a minimum the amount of compensation to be paid. They have added to their staffs a corps of doctors who are experts in their various lines. They have established clinics and in some cases own and control hospitals. They have established first-aid rooms and hospitals in connection with the various industrial plants throughout the Commonwealth, where a nurse continually and a doctor usually are in attendance.

Insurers seem to have left no stone unturned to give the injured employee the best possible treatment they can secure. His case is studied from the moment of the accident. If the case is a puzzling one to the doctor in attendance, consultations are held by various groups of doctors and the case is studied with the view towards finding the best possible method of treating it.

Although under our law the insurer is liable only for medical attention during the first two weeks, and for a longer period at the discretion of the Board, the insurance companies have had it brought home to them by hard experience that it is good economy, apart from the humanitarian aspect of it, to provide full medical attention. Most of the companies give this without any suggestion or interference from the Industrial Accident Board as to the unusualness of the case, provided the employee will place himself under the care of the insurance company. Insurers feel that it is a paying investment to expend vast sums of money on medical care. During the past year $2,250,000 was expended in Massachusetts for medical attention in the treatment of injured workmen. (Medical expenses the first year of the act were $414,195.42.)

In connection with the medical work being done by the insurance companies, the Industrial Accident Board has adopted a rule requesting a full medical report of all the treatment which has been or is being given an injured man after a certain length of time. These reports are studied by our medical adviser to see what, if anything, can be done to improve the treatment which is being accorded him to bring about his rehabilitation more rapidly. Many times injured men are brought in and referred to various experts whom the Board appoints as impartial examiners. The injured man is x-rayed, if necessary, and many times in the course of these impartial examinations he is examined by a half dozen doctors in various lines. If his case is an extremely difficult one he is referred to a hospital for admission and study by the entire staff of that hospital. After the study has been completed recommendations are made to the insurance company in a full written report. The insurer invariably carries out the suggestions and recommendations of the Industrial Accident Board. Subsequently, after these recommendations have been adopted, various reports are filed with the Board showing the progress of the case, so that we keep in touch continually with every case through the entire period of disability for which the man is receiving compensation. As a result of the methods adopted in Massachusetts, many marvelous feats of surgery have been performed and remarkable instances of rehabilitation are numerous.

As an example of how thoroughly our impartial physicians do their work I offer you the following report of an examination made in the case of an employee who had received a fairly serious injury to his hand and who was not responding to treatment as quickly as it was thought he should.

"I have carefully examined, February 6, 1923, the employee above named.

"I find sloughing granulating wounds of four partial fingers.

"The treatment indicated in this case is prolonged and very careful preliminary cleansing of the stumps of the four fingers and the whole remaining hand, then such partial amputations and plastic operations that all of his remaining hand may be preserved, together with the thumb. He will then have left eventually a useful thumb and some part or parts of stumps of fingers and palm of hand—which will be very useful in ordinary work.

"No amputations or plastic work should be attempted until the hand and stumps of fingers are thoroughly cleansed in gross and made as nearly as possible sterile. Any surgery done without great care in the above particulars might be attended by reinfections and serious consequences."

We have received the whole-hearted coöperation of the medical profession and the hospitals in Massachusetts. They have given us the very best that is in them in working out the problem of the rehabilitation of injured employees. All the medical work is being done and directed by Dr. Francis D. Donoghue, our medical adviser, who has been with the Board since the enactment of the act, and has given to this work the very best in him of the extraordinary knowledge and skill which he has obtained in his wide experience and education. While on this subject I cannot refrain from paying him tribute for the wonderful work he has done in this respect.

I must not neglect to discuss the importance of occupational therapy in conjunction with rehabilitation. Occupational therapy, properly applied, is the medium through which the desired result can be accomplished. It is the link connecting the bedside treatment with job placement. It nurses the stricken fibre of life back into fruition. On this subject a famous surgeon writes: "We learn to rely more and more upon therapeutic work for the physical, nervous and moral reconstruction which must follow medical treatment and without which much of the brilliant work of the operating room and the laboratory will be lost."

From the early stages the benefits of mechano—physio—and occupational therapy should be applied, looking forward to the best possible functional recovery for occupational adjustment. Naturally this should be done under the treatment of a competent medical man, familiar with the physical requirements demanded by industry, and he should eventually examine them with a view of making an appraisal of functions.

I could cite numerous instances of marvelous recoveries from serious injuries through the injured employees receiving the benefit of expert medical attention. While I do not want to weary this convention with stories of the many cases with which I have come in contact, I would like to tell of a few of them so that I might picture to you what happens when a cripple is restored to usefulness by proper medical care.

The most remarkable have been cases of so-called broken backs. I recall the case of William M..............., who was struck by a street derrick, knocked down and pinned to the ground. Jacks had to be utilized to lift the derrick from the employee's body. X-ray showed a crushing fracture of the second lumbar vertebra with dislocation forward of the first lumbar vertebra upon the second, also fracture of the transverse processes of the third lumbar vertebra on the left side. The lower portions of the employee's body were paralyzed and his case appeared to be a hopeless one. It was thought he would die as a result of the injury or remain a helpless cripple for the remainder of his life. Finally, a surgeon was appointed to study the case. Following his study an operation was performed, consisting in the removal of a quarter inch wedge of bone from the anterior portion of the tibia about eight inches in length, which was inserted into the split spinous processes of the 11th, 12th and four upper lumbar vertebrae (Albee Technique). Subsequently, the man left his bed and walked around with the aid of crutches. Later he used a cane and finally, after a remarkable convalescence, was able to walk without that help. I, personally, kept track of the case and found that the employee returned to his work within a year after the accident. His ability as a workman was later recognized and he was made a foreman and subsequently superintendent of a construction gang with materially increased wages. The benefits of that one case, both financially and economically, can hardly be estimated. Figuring roughly, the insurer probably saved in the neighborhood of $2500, taking into consideration the cost of medical attention and the compensation which had been paid the injured man before his return to work. He also was saved to industry and being a reliable man certainly is of benefit to it. Of course, we cannot overlook the benefit accruing to the little family of this workman. He is able to bring up his four children in comfort and educate them; they have not become the public charges they might have been had he not been restored to usefulness by competent medical attention.

I recall another case of a young man 19 years of age who received a very serious injury resulting in the crushing of his right hand. He was taken to a hospital where it was recommended that the hand be amputated. The young man demurred at this and would not give his consent. Later, he entered another hospital and received the same verdict. His case came before a member of the Board on the question of whether or not he was acting unreasonably in refusing to have the hand amputated. As a result of the conference, a famous surgeon in Boston was called into the case and, after examining and studying the injury, he said that he thought with a long period of treatment he could do something for the injured hand. The treatment was started and inside of six months the employee had a useful hand and after a long period of physiotherapy he went back to work. I have seen the hand and there is hardly any loss of function. If the hand had been amputated in the first instance, the insurer would have had to pay 50 additional weeks, at $10 a week, for the loss of the hand, and also disability compensation probably until the end of the compensation period. There was a saving of about $1500 as a result of providing this expert attention.

I remember another case, that of a middle aged man who had received an injury to his knee, tearing the ligaments and sinews so badly that it was decided finally that amputation of the leg at the knee was the only thing to be done. The man naturally was alarmed at the prospect of losing his leg and applied to a member of the Board to see if something could be done for him. As a result of the conference, this employee was turned over to a surgeon in Boston, who operated upon the leg, putting his expert knowledge and training into the work. Today, this man is walking without crutches or cane, with stiffness of the leg the only remnant of his injury. He returned to his old position and today is a happy, contented workman instead of a cripple with one leg.

Just before I left Massachusetts, I had a case in which medical attention was being given to an injured man. He was only entitled to fifteen weeks more compensation under the act. He was in an extremely bad condition and although the treatment accorded him has not brought about his recovery it has at least eased his pain during the years since his injury.

Many states give this question of medical care very little attention. I think it is absolutely wrong to limit the amount of medical attention which may be given in any one case. Medical attention should be unlimited. I have seen quite a few cases in Massachusetts where over $2,000 was expended for medical attention in each case. The insurers do it cheerfully as it is good economy. They are not always the gainers financially because sometimes, notwithstanding their efforts, they are not able to restore a man to usefulness, but at least they do the very best they can and that is all that can be expected of them.

From the experience gained in the administration of the Workmen's Compensation Act for twelve years, I would recommend most earnestly that every state, which has not already done so, take up very seriously the medical phase of the act and broaden its scope in every possible way.

BOOK REVIEWS

Diseases of the Nose, Throat and Ear—For Practitioners and Students. By A. Logan Turner, M.D., F.R.C.S.

The authors are leaders in their profession in Great Britain by reason of their scientific training, facile writing, intellectual attainment and large experience. Building on a similar and much smaller volume by Dr. W. G. Porter, who fell in the service of his country, and dedicating their work to his memory, they have added the results of modern investigation in sufficient detail not only to make this a valuable book for the beginner but to offer the advanced reader in concise form many interesting and valuable suggestions. It has the added merit of giving methods employed in Edinburgh, which in some instances vary from those practised in the United States.

There are 443 pages of closely printed matter, and the copious illustrations (mostly original) are small, contributing to economy in space. Emphasis is placed on anatomical, clinical and diagnostic details. Methods of treatment are explained. The technical operative details of the highly specialized departments of otolaryngology, such as bronchoscopy, esophagoscopy, laryngeal surgery, paranasal sinus operative work and the surgery of the mastoid and brain, are touched upon but not elaborated, for the scope of the work prevents.

Though there are five authors, each deals with so distinct a phase and each is so intimately associated with the others in his medical work that the book may be thought of, not as a compilation of scattered and diversified opinions, but as the combined product of a unified authoritative group.

A. Logan Turner and W. T. Gardner write the first two sections on the nose and accessory sinuses. The anatomy is given clearly and concisely. In the chapter on tonsil surgery by Douglas Guthrie ethyl chloride is recommended. Glover's inhaler and a Gibbs bag is used. With a death rate of one in 17,400 cases, this is considered a very safe general anesthesia. The guillotine method as devised by Whillis and Pybus in 1910 (one year after Sluder brought forward his alveolar eminence method) is the one generally used in this country. The patient lies flat, with the head lowered or the shoulders raised. The dissection method is used more in London, especially when the guillotine is not applicable, and local anesthesia is preferred. Guthrie finds naso-pharyngeal fibromata rare, locally malignant but not metastatic, and with a grave prognosis because operative removal is so difficult and recurrence so likely. The use of radium or deep x-ray is not mentioned.

Section IV on the larynx is by J. D. Lithgow. The colored pictures showing laryngeal pathology are very good. Treatment is stressed here as it is throughout the book. The different laryngeal motor paralyses are carefully explained. Laryngeal tumors and their operative removal are touched upon. The value of x-rays or radium is considered at best to be but palliative in malignant forms.

The next two chapters, by G. Ewart Martin, are devoted to peroral endoscopy and bronchial and esophageal affections. He finds practically no contraindications to bronchoscopy, excepting in cases of aneurysm, pulmonary tuberculosis, mitral incompetence and advanced arteriosclerosis.

The sixth and last section, of 145 pages, is on the ear and is written by J. S. Fraser. After a careful consideration of the anatomy, twelve very helpful pages are devoted to the vestibular apparatus. Then follows a discussion of external and middle ear and mastoid diseases. Emphasis is placed on intracranial complications and affections of the labyrinth. His intimate knowledge of the microscopical pathology is reflected in these pages.

An appendix giving favorite medications and a detailed index close what will prove a valuable and up-to-date addition to our otolaryngological text-books.

Basal Metabolism in Health and Disease. By Eugene F. DuBois. Published by Lea & Febiger, Philadelphia and New York. 1924. 372 pages. Price, $4.75.

DuBois has been the leader in the recent advances in clinical calorimetry in this country. It is, therefore, of particular interest to read a summary of the subject written by him, and this volume is as carefully and accurately done as everything else he has written. It is indeed an excellent short summary of our present knowledge of basal metabolism in health and disease—in which all important facts are recorded and well discussed.

In reading the book one only wishes that the author had been a little freer in his criticisms, and had so made the book a little longer. It is by far the best summary of this subject which has appeared, and will probably long remain the authoritative book in this field, particularly if future editions contain the further advances which will be made in this science of clinical medicine. It is a book all progressive doctors and medical students should read.

Volumetric Analysis. By Francis Sutton. Eleventh edition, revised by W. Lincoln Sutton and Alfred E. Johnson. Philadelphia: P. Blakiston's Sons & Co. 1924.

A book which has reached the eleventh edition and has been a standard since 1863 scarcely needs an introduction. However, this is the first revision since 1911 and a number of changes have been made. Among the most important of these changes is the revised chapter on gas, including micro-gas analysis, in which the methods of Haldane, now so widely used in biological work, are described.

Although the subjects in which the medical profession is most interested receive comparatively little attention, the work being planned chiefly for the use of commercial analysts, anyone making volumetric analyses will find in it many useful facts and suggestions, many valuable tables, etc.

Clinical Studies in Epilepsy. By Donald Fraser, M.D. Pp. 238. New York: William Wood & Co. 1924.

Dr. Fraser, who is consulting physician at the Paisley District Mental Hospital, England, has written a small book, largely made up of clinical notes on cases of epilepsy under his observation. One case report, given in detail, covers a period of forty years and clearly illustrates the marked periodicity of this disease as well as the various types of treatment suggested by the medical profession, and others, in the last two decades. The author discusses, in relation to his cases, the possible causative factors in idiopathic epilepsy as follows: Vaso-constriction, the cerebrospinal reflex, endocrine glands, traumatism, tumor, tuberculosis, syphilis, and special conditions affecting the initiation and development of the epileptic movement. He quotes frequently from Hughlings Jackson. The author's conclusions are difficult to find, being scattered throughout the book. Apparently he accepts the Hughlings Jackson hypothesis of vascular changes in the pial vessels, perhaps due to a toxin. The book, while of interest to the special student of epilepsy, adds little to our knowledge of either the cause or the treatment of this distressing syndrome.

Studies from the Rockefeller Institute for Medical Research. Reprints. Volumes XLVIII and XLIX.

Two large volumes containing the results of investigations conducted at the Rockefeller Institute or elsewhere under its grants. These are collections of papers which have first been printed in a variety of journals, so that these volumes contain only reprints of work recently reported. Many of the articles are of great interest, and they give an excellent impression of the varied activities fostered at the Institute.

Materia Medica for Nurses. By A. L. MUIRHEAD, M.D., Late Professor of Pharmacology, Creighton Medical College, Omaha, Nebraska, and EDITH P. BRODIE, A.B., R.N., Instructor in Materia Medica and Therapeutics, Washington University School of Nursing, St. Louis, Missouri. Second edition. St. Louis: C. V. Mosby Company. 1924.

The principles embodied in this little book are valuable for a nurse's training in that they contain practical information concerning the methods of computing doses and making up solutions. A number of the more useful drugs are discussed briefly as to action and incompatibilities, and antidotes are given. There are some important omissions, such as mercurochrome; it is also worthy of note that so important a therapeutic agent as cod liver oil is dismissed with a paragraph mentioning its unpleasant taste and an unusual method of administering it in the treatment of malnutrition in children, and entirely ignoring its extreme value in the prevention and treatment of rickets.

Safeguarding Children's Nerves. A Handbook of Mental Hygiene. By JAMES J. WALSH, M.D., PH.D., Sc.D., Professor of Physiological Psychology, Cathedral College, New York, and JOHN A. FOOTE, M.D., Professor of Diseases of Children, Georgetown University Medical School, Washington, D. C. With a Foreword by Honorable Herbert Hoover. Philadelphia and London: J. B. Lippincott Company.

"The irritable or 'nervous' child is a source of worry and anxiety that creates a vicious circle of solicitude in the family," in the words of the authors. 'Yet the problem of preventing at least the worst developments of this family nightmare is not difficult if only faced from the beginning and met with the knowledge that is now available as to training in self-control, and such discipline of mental and physical powers as will assure that most precious of all possessions for life, 'a healthy mind in a healthy body.'"

The human nervous system is unquestionably having a hard time to keep up with the rapidly increasing complexity of its environment. This fact is being generally recognized even while the causes of maladjustments, mostly arising in childhood, and the proper methods of overcoming them are still somewhat obscure to the majority of us. The new science of mental hygiene is becoming a very popular, and is certainly a very useful, branch of medicine. Dr. Walsh and Dr. Foote show that they thoroughly appreciate its value and its application.

All pediatricians of the past and many of the present recognize a type of infant that is almost sure to become a feeding problem. Hitherto this problem has always been attacked from the feeding end, and various methods and mixtures have been tried until eventually the case straightens out. These authors point to the fact that it is not an unsuitable food that is disagreeing with an infant; it is an abnormal infant that is disagreeing with its food. The congenitally nervous infant is the earliest type of a hypertonic organism that is out of tune with its surroundings.

"The Nervous Child," "The Spoiled Child" and "Misunderstood Children" are chapter headings. Under "The Bogy of Heredity" the authors attempt mercifully to dispel the hopelessly fatalistic attitude that so many individuals have come to adopt toward the inheritance of personal characteristics. It is well to remember that insanity, for instance, tends to disappear through succeeding generations, and that feeble-mindedness is a recurring character.

Various interesting and valuable points brought out by the authors are the danger of moving pictures, both as an undesirable mental stimulant and as a deterrent from proper recreation, and the need for discipline as opposed to those who believe that the child's character should be allowed to develop unhampered. In the face of ultramodernity it is to say the least refreshing to find some who dare recall the adage of sparing the rod and spoiling the child. "Parental laxity," we are told, "is not love, but very often laziness and lack of character."

Chapters on "Habits," "Dreads and Dislikes," "Backward Children" and "Defective Children" bring the volume to its close. Some looseness of diction and a wealth of split infinitives can be forgiven in authors who abandon abstruse theory and talk in terms of common sense.

An Outline of Endocrinology. W. M. CROFTON. Published by Wm. Wood & Co. 1924. 126 pages. Price, $2.25.

This outline is a short summary of a difficult subject, which is well written and concise and therefore of considerable help to medical students, and practitioners who desire such information. The book is an outgrowth of a series of lectures, and contains facts chosen from the vast amount of literature which has accumulated in the field of the internal secretions. There is no attempt to make the book complete or to include all that is important, and, like most text-books, it is several years behind the current medical literature. One fundamental criticism seems justified, for, throughout the volume, there is a mixing of facts learned in the laboratory and inferences gleaned from clinical pictures. By the method of presentation it is often difficult to evaluate statements, for there is a great difference in the relative importance of laboratory observations and assumptions based on isolated cases of disease as seen in the clinic.

Mikrochemisches Praktikum. Eine Anleitung zur Ausführung der wichtigsten mikrochemischen Handgriffe, Reaktionen und Bestimmungen mit Ausnahme der quantitativen organischen Mikroanalyse. VON FRIEDRICH EMICH. Published by J. F. Bergmann, Munich. 1924. 174 pages. Price, $1.55.

A small text-book describing various methods, largely German, of micro-chemical analyses. These are largely for inorganic salts, though there are also many methods of organic analyses. The large field of biological analyses by micro methods, as developed by such men as Folin, is not fully covered in this book. As a text-book containing many helpful hints and many methods of value, however, this small volume has a definite field.

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 11021

MEDICAL DEPARTMENT

First entry. A man of thirty-four was brought to the Emergency Ward October 26 semi-conscious, with stertorous and labored breathing and very alcoholic breath. No history was obtained. His breathing became more and more labored and struggling. He tossed a good deal. There was depression of the suprasternal notch. He became black.

P. E. Showed no evidence of injury and no abnormalities except systolic B. P. 190.

Tracheotomy was done at once. He had stopped breathing, but came back quickly. October 29 he was mentally clear, but remembered nothing of the night when he came in. He breathed freely through the tracheotomy tube. There was occasional cough. He was discharged that day to the Eye and Ear Infirmary.

History of interval. At the Infirmary he was comfortable, but had some difficulty in getting enough air through the tracheal tube. He said that he had felt perfectly well up to the time of entering the Massachusetts General Hospital. Later he gave a history of falling and injuring his throat. No foreign body was seen. The larynx was reddened and edematous and made very slow improvement. He ran a moderate septic temperature. November 6 it rose to 104°. A medical consultant found dullness in the right chest. The leucocyte count was 23,000.

Second entry, November 9. P. E. A rather plethoric man, only mildly dyspneic, unable to speak so as to be understood. Throat generally reddened. Tonsils slightly enlarged. Questionable flexibility of the spine in the lumbar region. *Heart* normal. *B. P.* 135/90. *Lungs.* Examination unsatisfactory because of the tracheotomy. Dullness with no apparent change in breath sounds at the right base. *Abdomen* normal. *Extremities.* Slight tremor. *Rectal examination* not done. *Pupils* and *reflexes* normal.

T. 104.8°-100.4°. *P.* 83-120. *R.* 24-55. *Urine.* Normal amount on the two occasions recorded, sp. gr. 1.020, a very slight trace of albumin at one of two examinations, occasional leucocytes at one. *Blood.* Hgb. 95%, leucocytes 20,000-50,000, polynuclears 87%, reds normal. *Wassermann* negative. *Sputum.* Pneumococcus Type IV.

The patient did not look so ill as would have been expected. November 10 he complained of pain in the right side. He coughed up thick tenacious sputum, slightly blood streaked, through the tracheotomy tube, which easily filled up. Next day Dr. White found distant bronchial breathing in addition to dullness at the right base. A not entirely satisfactory X-ray film of the chest showed a triangular area of increased density in the right lower midchest quite suggestive of consolidation. The outline and position of the diaphragm were not abnormal. The costophrenic angles were clear. There was no definite evidence of fluid.

November 13 a throat consultant reported, "Left vocal cord fixed. Considerable edema of false cord and left arytenoid. Punched-out area of right cord, lower third. Do not advise removal of tube at the present time."

The patient gradually grew weaker and had more and more difficulty in breathing. The tube became plugged. November 13 it was changed. He weakened rapidly and died that night.

DISCUSSION

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

"He became black" is a positive statement which comes in a little suddenly. I suppose it means that he became tremendously cyanosed.

Why did they discharge him to the Eye and Ear Infirmary? Neither his eyes nor ears had been complained of. Presumably because they thought there was something in his throat which this tracheotomy relieved—what, we have no hint.

MISS PAINTER: It was thought that he was intoxicated at the time he was brought in first.

NOTES ON THE PHYSICAL EXAMINATION

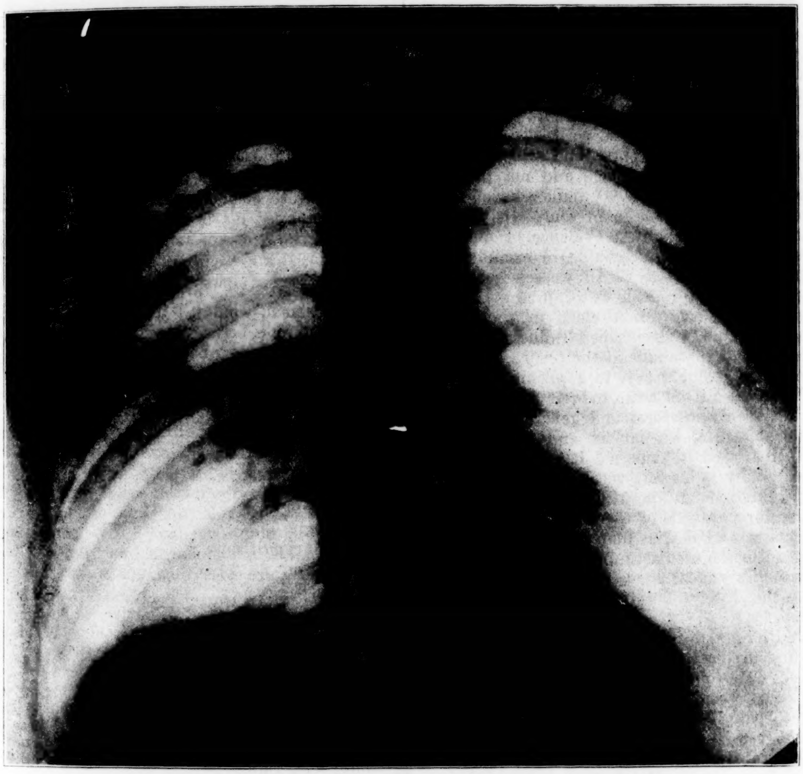
DR. CABOT: Tracheotomy spoils all the lung signs. We cannot tell what we are hearing. Undoubtedly they are suspecting a pneumonia or an empyema.

The finding of the Type IV Pneumococcus does not in any way prove that he has pneumonia. We get that type in sputa of all sorts.

The pain was presumably in the right side of the chest, because it was at the right base that dullness was found.

This is of course exactly the sort of case in which if we can get a portable machine and can have X-rays taken in the ward without disturbing the man at all it is of great value. The ordinary methods of examination are very much handicapped on account of the tracheotomy tube.

In relation to treatment it does not make a great deal of difference whether we are sure that he has pneumonia or not. For Type IV we have no special treatment. In relation to prognosis it is, of course, important to know.


Shows a triangular area of increased density in the right lower midchest quite suggestive of consolidation. The outline and position of the diaphragm are not abnormal. The costophrenic angles are clear. There is no definite evidence of fluid. The plate is not entirely satisfactory.

The costophrenic angles would not be clear with fluid unless it was encapsulated, and it takes time to encapsulate fluid. This is rather a short period for it.

In the first detailed account of the throat that we have the left vocal cord is presumably paralyzed.

DIFFERENTIAL DIAGNOSIS

This is a blind case. Granting that he has pneumonia, which I think the X-ray gives us good reason to believe, that is certainly not all he had. He had something obstructing or pressing on his upper respiratory passages. The two things that come first to mind are (1) deep cervical sepsis, the thing called Ludwig's angina, a sepsis working down into the deeper tissues of the throat, often causing a great deal of dyspnea and cyanosis with fever and leucocytosis, as he has here. Ordinarily however a deep cervical sepsis gives external evidence, tenderness, redness, swelling on the outside of the neck. I do not understand that there is anything of that kind there. From the record the trouble seems to be all inside and none outside. And in the cases that I remember of deep cervical sepsis there has been something outside as well as in.

Granting that he was drunk in the beginning, he probably was something else besides drunk. He certainly did not fall unconscious and get black in the face from pneumonia or alcoholism. That, so far as I see, calls for some obstruction in the upper air passages. Can that obstruction have been all in the larynx? Yes, so far as I know. One vocal cord we know is paralyzed. The other has been swollen and we can well imagine enough obstruction to cause deep cyanosis and through that total unconsciousness.

What causes paralysis of the left vocal cord often? When we find one vocal cord paralyzed and that the left, what is the thing we should say?

A PHYSICIAN: Aneurism.

DR. CABOT: We certainly have to think of that. Alcoholics are a little more likely to have syphilis than other people. Syphilis often hits the larynx. The ulcer on the left vocal cord may be syphilitic for all we know, and if he has an aneurism, that with its effects might account for the paralyzed cord. But we have had an X-ray of the chest, and although it was directed towards the lungs and not primarily towards the heart, it would have shown aneurism if it had been present. In the plate we see not the slightest evidence of aneurism. We certainly should be able to exclude aneurism from this plate. All we see is the area described in the report. The right lung is clear above, clear below, not clear in the middle, where we see wedge-shaped dulness, with the thick end of the wedge at the pleura, and corresponding, I should say, to a pneumonia.

What does it all come to? It comes to pneumonia with some local and general sepsis, that sepsis affecting his larynx and perhaps other tissues around it, though I have no evidence of that. Why is his left vocal cord paralyzed? I don't know. I know no regular, recognized cause for paralysis of the left vocal cord except aneurism and mediastinal tumors. Hodgkin's disease in the mediastinum can press on the recurrent laryngeal nerve so as to give paralysis. But the X-ray, I think, excludes mediastinal tumor.

A PHYSICIAN: Does that history of fall with injury to the throat suggest anything?

DR. CABOT: That does not interest me at all. I cannot imagine a fall with serious injury to the throat. No external injury that I know of, without wound—and there is no wound—could paralyze his vocal cord.

A PHYSICIAN: Could it cause a hematoma and that press on the laryngeal nerve?

DR. CABOT: I cannot say no. It seems, though, as if somebody would have got evidence of that, either externally or internally.

DR. YOUNG: Would the pressure of the tracheal tube itself do such a thing as that?

DR. CABOT: I have never heard of it. It is a new idea to me. Moreover he evidently had something blocking his larynx before he had the tube in.

A PHYSICIAN: Did he have diphtheria?

DR. CABOT: That is a good question. They certainly had a good chance to find it out if he had. I should say that with the quality of the examinations that we must suppose he had, no, he did not have diphtheria. Of course blocking of respiration from diphtheria is common, but when we have as good a chance to see as we had here we should be able to recognize it.

I cannot go any further. My impression is that his pneumonia was the largest element in his death, that the other lesions woud probably have got well if he had not had the pneumonia, and that the tracheotomy could later have been closed.

I do not see why anything should be found outside the organs referred to, that is, the lungs and the throat.

DR. YOUNG: Did you come to any conclusion as to why he was plugged up when he first came in?

DR. CABOT: I suppose he had something in his larynx then. He probably had a septic laryngitis with alcoholism, and the combination of these stopped his breathing. It is not very satisfactory. I do not remember any case like it.

A PHYSICIAN: A foreign body would have been mentioned, wouldn't it?

DR. CABOT: Yes. They specifically say there was no foreign body.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the larynx.
Lobar pneumonia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Pneumonia.
Septicemia.
Septic laryngitis.
Alcoholism.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Ulcer of larynx.
Purulent tracheitis and bronchitis.

2. *Secondary or terminal lesions*

Septicemia, streptococcus hemolyticus.
Empyema, right.
Slight fibrinopurulent pleuritis, left.
Acute pericarditis.
Acute general peritonitis.
Compression atelectasis.

DR. RICHARDSON: The peritoneal cavity contained a small amount of thin purulent fluid, and there was a thin fibrinopurulent layer of exudate over the peritoneum rather generally. Cover-glass from the exudate in the peritoneal cavity showed streptococci.

The diaphragm on the right was at the fourth interspace, on the left at the fifth interspace.

The right pleural cavity contained at least 1500 c.c. of pus, and the lower half of the right lung was collapsed because of the pus—compression atelectasis. On the left side there was about 200 c.c. of purulent fluid.

The pericardium contained a small amount of purulent fluid, and there were areas of ecchymosis scattered over the surface,—acute pericarditis. The heart weighed 385 grams,—a large heart, but he was a large muscular man. The circulatory apparatus generally was frankly negative.

In the larynx there was an extensive area of ulceration which ran around the wall between the base of the epiglottis and the cords and hit the posterior third of the cords. In back the ulceration extended down for a short distance below the cords. The surface of this ulcer was necrotic, and at one place there was a small depression which reached the wall of the esophagus but did not penetrate it. The perilaryngeal tissues showed some purulent infiltration which extended down and into the pleura. Macroscopically there was no evidence of any definite masses or nodules of new growth. Microscopically it is a chronic ulceration with necrosis and suppuration. There is no evidence of carcinoma.

Culture from the heart blood showed a profuse growth of streptococcus hemolyticus.

DR. CABOT: Apparently we could not have a more sharp discrepancy between the ante- and the post-mortem diagnoses. But I do not think it is quite so sharp as it sounds. In part it is a difference between different periods of observation. There was no possibility of free empyma at the time this X-ray was taken. It shows perfectly clear costophrenic angles. Could he have had a pneumonia which resolved entirely before the empyema came on? I do not know enough to say. The lung was clear above and below the dull area. The lung evidently has not collapsed. There was certainly no fluid at the base when the X-ray was taken. If he did not have pneumonia he must have had an encapsulated interlobar empyema which later broke out and affected the whole pleura.

DR. RICHARDSON: I think your explanation is right, that it hit at one place and then extended, and finally the empyema was established and pushed up the lung.

DR. CABOT: The same process may have occurred in the other lung. At the time of the X-ray there certainly was no fluid there. I suppose the pericarditis and peritonitis came after the period we recorded. But I think it is very interesting that the peritonitis should have been altogether symptomless. Every time this happens it surprises us afresh to have a patient show no reaction to his peritonitis in the way of tension, spasm, etc., especially when he has had no operation and has been on his feet until fairly recently.

DR. YOUNG: What is the date of that X-ray? Wasn't it taken fairly recently?

DR. CABOT: On the 11th of November.

DR. YOUNG: He died on the 14th. It is a very quick change.

DR. CABOT: It was three days before his death apparently. I think it is quite possible that within three days the lesions could have changed so much. When empyema gets going it goes very fast.

A PHYSICIAN: Can that have been a bronchial tree plugged with pus?

DR. CABOT: I don't think so. I never heard of any such thing.

Where should we suppose all this sepsis started? Is there any reason why we should not suppose that it started in the septic process in the larynx, got into the blood, and was from there distributed to all these membranes?

DR. RICHARDSON: There was some purulent infiltration down along the pharyngeal tissues.

DR. CABOT: We have no reason to treat this laryngitis as a terminal phenomenon. One of the things he had in the beginning was this laryngitis. If we accept that, the sepsis found in his blood, pericardium, peritoneum and both pleurae started there. I suppose that his original unconsciousness and tremendous cyanosis were due to laryngeal block, due to edema as a part of this ulcerative process. There was nothing in the deeper tissues of the neck?

DR. RICHARDSON: Only some purulent infiltration close around the necrotic wall of the larynx. At one point there was a necrotic area which had reached the wall of the esophagus but had not extended into the wall.

DR. CABOT: I am sorry we have no X-ray expert to interpret this plate, because I have never seen an interlobar empyema confined between the lobes that gave that picture. Yet I do not know what else to call it.

NOTE BY DR. G. W. HOLMES

I agree entirely with Dr. Cabot's explanation of the discrepancy between the X-ray appearances and the autopsy findings. I feel sure there was no free fluid in the pleural cavity at the time the X-ray was taken.

CASE 11022

MEDICAL DEPARTMENT

An American court officer of fifty-two was sent from the Emergency Ward at one a. m., October 24. The only history was obtained from his daughter.

P. I. For four or five days before admission he had had some trouble with speech, but otherwise had been in fairly good condition. A short time before being brought to the Emergency Ward he was found unconscious, having apparently had a sudden attack.

P. E. A somewhat obese man, unconscious, with stertorous breathing, moving the left arm and leg but not the right. Incontinent of urine and feces. Tongue and buccal mucous membranes bitten on the right side. Apex impulse of the *heart* in the fifth interspace 14 cm. from midsternum, 6 cm. outside the midclavicular line; supracardiac dullness 7½ cm. right border 3½ cm. to the right. Sounds and action normal. No murmurs. Pulses normal. Radials hardened, palpable. Left temporal tortuous. *B. P.* 230/135-300/195. *Lungs.* Both chests largely obscured by noisy breathing. A few moist râles below the angle of the left scapula. ***Abdomen***, *extremities* and *pupils* normal. ***Neurological examination.*** It was impossible to tell much about the face. The right upper eyelid seemed to have less tone than the left. Nothing could be told about the tongue. Absent triceps, biceps and supinator on the right, absent biceps and supinator on the left. Flaccid paralysis of the right arm and leg. Knee-jerk absent on the right, present on the left. Ankle-jerks normal. Babinski negative. Practically no plantar reflex on the right. Oppenheim not definitely positive on either side. No clonus or Kernig. Right cremasteric absent, left questionably positive.

T. 98°-100.2°, *P.* 100-110, *R.* normal, *urine* and *blood* not recorded.

The patient did not regain consciousness. At ten o'clock the morning of admission he died.

DISCUSSION

BY DR. MAURICE FREMONT-SMITH

NOTES ON THE PHYSICAL EXAMINATION

DR. FREMONT-SMITH: Is this blood pressure right and left arms, or is it two different records?

MISS PAINTER: It is two different records.

DR. FREMONT-SMITH: I have never seen a diastolic pressure as high as 195.

DIFFERENTIAL DIAGNOSIS

In the first place we must think of uremia, which, although it does not usually, does sometimes do exactly this,—begins with sudden unconsciousness. We do not get paralysis in uremia although we must remember that with a chronic nephritic with high blood pressure one may run into one of these cerebral crises with spasm of the vessel.

Of course the great probability here is that this man four days before entrance had a slight cerebral hemorrhage either hitting the brain area of the tongue or involving Broca's area so that he had difficulty in finding his words, an aphasia; that this was followed after four days by a second profuse hemorrhage on the left side giving him flaccid paralysis on the right. Of course we should expect a spastic paralysis with increased reflexes, because the cerebral inhibition is cut off. Therefore the muscular tone and reflexes should be increased. Directly after a hemorrhage, however, and for a variable time, I should say rarely as long as a week or ten days, one may have complete disappearance of all reflexes and a flaccid paralytic condition.

There is everything here to make us think of cerebral hemorrhage,—his blood pressure of 300, his tortuous arteries. We have to remember, however, that a brain abscess might do this; a brain tumor lying dormant with a hemorrhage occurring in the tumor will give this same picture.

A PHYSICIAN: A thrombus?

DR. FREMONT-SMITH: A thrombus or embolus would give us exactly the same picture. Of course in the heart we heard no murmurs, but that does not mean that there may not be a thrombus in the auricle.

Malignancy has to be thought of, malignancy anywhere, with metastasis to the brain, metastasis for a time silent, and perhaps a hemorrhage into the metastasis or caused by the metastasis. But I believe we have a simple case of cerebral hemorrhage.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Cerebral hemorrhage.
Right hemiplegia.
Arteriosclerosis.
Hypertension.

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Cerebral hemorrhage.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Arteriosclerosis of the vessels of Willis.
Cerebral hemorrhage.

2. *Secondary or terminal lesions*

Hypertrophy and dilatation of the heart.

DR. RICHARDSON: Rigor mortis was generally well marked except in the forearm and hand of the right upper extremity. We have noticed a number of times here that on the paralyzed side, or the side said clinically to have been paralyzed, rigor mortis is not so marked as it is on the good side. We try to remember to look for that, and when we do it has seemed to be so.

The heart weighed 660 grams. That is a considerably enlarged heart, even for him. The valves were frankly negative. There was only slight dilatation. The myocardium was thick, four mm. on the right, fifteen on the left. There was only a slight amount of arteriosclerosis, nothing whatever to account for the size of the heart. Of course it falls into the group which clinically is called hypertension.

Below the clavicles I found no cause for death except the hypertrophied and dilated heart. And if we could not have examined the head in this case we should simply not have known.

The vessels of Willis and the branches near and remote showed considerable arteriosclerosis, and on the left side, beginning in the posterior region of the left frontal lobe and extending backward as far as the posterior third of the left optic thalamus and to the right for a short distance in the right cerebral hemisphere, was a large mass of blood clot which had ruptured into the left lateral ventricle, and there was, of course, considerable blood and blood clot in the other ventricles.

CASE 11023

Surgical Department

First entry. An Irish housewife of forty-two entered March 22, twenty-two years before her second admission. She was in the hospital eighteen days with an attack of "croupous" pneumonia.

Second entry. November 6, twenty-two years later, she came for relief of vomiting, anorexia and weakness. She was now widowed and employed in a factory.

F. H. She knew nothing of the causes of death in her family.

Habits. She formerly used to take at least a bottle of beer or whiskey a day. Lately she had used no alcohol.

P. H. She formerly had occasional "rheumatism"—never acute symptoms, and none in the past few years, except that her knees were now stiff. She had had a few sore throats. She had had dull constant pain in the right arm lasting a few weeks ("neuritis"). Ever since her attack of pneumonia she had been subject to colds and bronchial trouble. Five years before admission she had a mild attack of influenza. She had had several falls, bumping her head and once breaking her arm. She formerly had severe frontal headaches with nausea and vomiting occurring without known cause once or twice a month and lasting three days. Until three weeks ago she had had frequent urination, passing small amounts. She formerly had prolapse of the uterus necessitating a rubber ring.

P. I. Five years ago she lost appetite, and found that if she ate meat, cabbage, bananas, etc., she had belching of gas and indefinite gastric discomfort. She had practically restricted her diet to cereals, soup, potato, milk, tea, bread, and an occasional egg. She lost not over ten pounds. She began to have occasional periods of weakness, feeling faint and dizzy for about a minute perhaps only once a month. For five years she had no breakfast and little at the other meals. After eating much she had distension. Until two weeks ago she had been troubled a good deal with gas and belching relieved by soda. Several times she had to leave work for periods of a week to two months when feeling sick or run down, or when she had a heavy cold. Nine months ago varicose veins developed. (At her first entry she gave a history of varicose veins two years earlier.) Seven months ago she was laid off from work and felt too old and weak to try to look for work again. She lived mostly on bread and milk. Eight weeks ago the condition, which had remained about the same for five years, became worse. Vomiting had been a prominent symptom. After eating she would have indefinite gastric discomfort—no real pain—and a desire to get food off her stomach. The vomitus usually consisted largely of food; bitter, burning, yellow or green, never bloody or coffee grounds. The vomiting was usually some time after eating, perhaps eight hours. This occurred about three times a week. Two weeks ago she became much worse, vomiting every day nearly everything eaten. She had eaten very little and had lost some strength. Her bowels, usually regular, had been irregular, requiring cathartics. Two weeks ago and again three days ago she had tarry stools.

Records of the Out-Patient Department September 1, fourteen months before admission. Complaint, stomach pain and gas for a year, worse after meals; very weak. Examination negative except for ptotic abdomen. A belt was fitted. September 29, a year later. History covered above. Lungs. Slight hyperresonance all over the chest. The right lower lobe crepitant on inspiration. Chest slightly emphysematous. Abdomen negative. B. P. 160/100. X-ray. Stomach contained approximately half the meal at six hours. An appreciable residue was present at twenty-four hours. Peristalsis irregular and at times deep, suggesting obstruction. Pyloric region irregular and narrow. Barium passed through it with difficulty. First portion of duodenum never filled with good outline. Remainder of tract not remarkable. October 16 Wassermann negative.

P. E. Fairly well nourished. Teeth all gone. Apex impulse of the *heart* not found. Action slow, regular. Sounds not of good quality. First sound reduplicated at apex. A_2 accentuated and snapping. No murmurs. Artery walls slightly palpable and tortuous. *B. P.* 190/100.

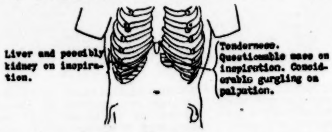
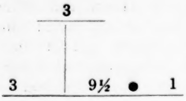
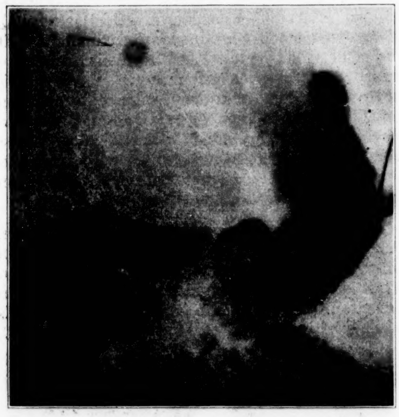

Liver and possibly kidney on inspiration.
Tenderness. Questionable mass on inspiration. Considerable gurgling on palpation.

FIGURE I.



Lungs normal. *Abdomen.* See Figure I. *Pelvic examination.* Marked cystocele and moderate rectocele. Cervix not felt (drawn up). Os slightly enlarged and surrounded by old scar tissue. Resistance (old adhesions in vault), more on the left, with some tenderness. Fundus retroverted. *Rectal examination* negative. *Extremities.* Varicose veins in the lower legs. *Pupils.* Left slightly irregular. Reactions normal. *Reflexes* normal.

T. 99.9°-97.1°. *P.* 88-60. *R.* 16-23. *Urine.* Amount normal on the one occasion recorded, sp. gr. 1.004-1.010, alkaline at one of two examinations, leucocytes at both, no albumin. *Blood.* Hgb. 75%-65%, leucocytes 10,000-12,800, polynuclears 58%, reds 3,500,000-4,200,000, slight achromia, platelets normal. *Wassermann* negative. *Stools* negative. *Gastric analysis* November 7. Tube very easily passed. *Fasting contents.* 50 c.c. clear transparent mucoid fluid with a slight yellowish tint and very few particles in suspension. No free HCl. Total acid 15 per 100 c.c. Guaiac negative. No evidence of stasis. *Test meal.* 75 c.c. cloudy opalescent fluid with food particles. Free HCl 10 per 100 c.c. Total acid 20. Guaiac test and microscopical examinations negative. November 11, sixteen hours after a meal of spinach, prunes, bread and tea, *fasting contents* 50 c.c. of slightly cloudy faintly yellowish fluid with very few shreds. Findings practically as before. *Test meal.* 50 c.c. of cloudy slightly yellowish fluid with gross food particles in abundance. Finding as before. *X-ray* findings practically the same as at the first observation except that there was less evidence of obstruction, there being only a small residue present at six hours. *Medical consultation* November 7. . . . . Operation definitely indicated. *Surgical consultation* November 7. Under the circumstances an exploratory operation would seem to be indicated. November 14. "With the new X-ray and tube findings and the gain in weight since entering the hospital I think it would be better to continue with medical treatment provided she can be kept under observation at definite intervals."



November 12. Findings practically the same as at the first observation except that there was less evidence of obstruction, there being only a small residue at six hours. (At the first examination the pyloric region was irregular and narrow and the first portion of the duodenum never filled with good outline.)

Orders. November 6. Five meal gastric diet November 8. Soft solid diet. November 20. Soda bromide gr. xx.

By November 10 the patient was sitting up and felt well. She had not vomited since entrance. November 20 she was discharged.

History of interval. Record of the Out-Patient Department December 4. She had not followed diet and had been under-eating. December 2 she had vomiting and gaseous eructations. January 29 she had the left fibula malleolus fractured in a street car accident. She had vomited five times since the last visit in spite of rigid adherence to diet.

Third entry. February 25, three months after the last discharge. As a result of the accident January 29 she lost much sleep and became irregular about meals. Three weeks following the injury her appetite was very poor. Four days ago she vomited once, whitish fluid, following a meal of milk two hours earlier. Two days ago she vomited similar material just before going to bed. The day before admission she vomited three times, milk taken an hour earlier. She vomited once the day of admission. There had been no pain before or after vomiting, no hematemesis, tarry or coffee ground stools. She had been gaining weight since leaving the hospital and had noticed no loss of strength.

P. E. (As before except as noted.) *Pupils.* Sluggish reactions. *B. P.* 160/105. *Abdomen* as shown in Figure II. Before operation *T.* 97.6°-99°, *P.* 65-90, *R.* 12-26. *Urine* not recorded. *Blood.* Hgb. 80%, leucocytes 10,200, polynuclears 61%, reds normal except for slight achromia, platelets large and increased. *Wassermann* negative. *Stool* negative.

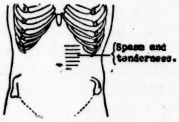


FIGURE II.

A surgical and two medical consultants agreed in advising operation. February 28 it was performed. The patient was in fair post-operative condition until the following morning, when the pulse rose to 120 and became much weaker, and edema of the lungs developed. That night the temperature rose to 101°. In spite of all efforts at cardiac stimulation she went rapidly downhill and died, apparently of bronchopneumonia, March 1.

DISCUSSION

BY DR. HUGH CABOT

I do not see that the "croupous" pneumonia of her first entry twenty-two years ago bears at all upon her present condition. Her habits of the past in regard to alcohol do seem to me important and might at least be suspected of having contributed to make her somewhat older than her years. Her previous history contains nothing that clearly bears upon her present illness, though it may well be that her falls were not unrelated to alcohol.

The condition with which it seems to me we are concerned apparently dates back five years and was characterized by the picture which one might call chronic dyspepsia. There is no history of a direct relation between this dyspepsia and the taking of food, particularly no clean-cut picture such as one occasionally sees with gastric or duodenal ulcer. The attacks of weakness, faintness and dizziness may be due simply to her restricted diet, but may also be due to cardiovascular disturbances. On the whole she seems to have been scaling down her food during this period and is now not getting enough to keep her in good condition. Quite recently there has appeared a definite history of vomiting apparently of only a few months' duration, but the symptoms have been progressive and the vomiting has now become a very serious symptom.

At the time she was seen in the Out-Patient Department some fourteen months before admission they did not find any very definite trouble and regarded her difficulties as largely due to ptosis. A year later, however, very definite abnormalities were found, some of which may perhaps have been present at the earlier date. There was apparently clear evidence of some hypertension, definite emphysema and probably some chronic bronchitis. The examination of the stomach showed clear evidence of obstruction.

A more complete examination made at her admission somewhat over a month later shows definite evidence of arteriosclerosis with hypertrophy of the heart and rather more hypertension than at the previous examination. The examination of the abdomen shows that the liver can be felt, but it is perhaps nothing more than a low liver. Beyond a rather low specific gravity there is no abnormality of the urine, but it is at least suggested that she may have definite arteriosclerotic nephritis. She also had a moderate degree of anemia. The examination of the stomach contents shows a rather low acidity and definite evidence of obstruction, but does not help us much in a differential diagnosis.

The chief interest here centres about the question of the nature of the stomach lesion. She is clearly a woman quite as old as her years, with definite arteriosclerosis and hypertension. She has well marked obstruction to the outlet of the stomach, and the important question is whether this should be regarded as cancer or as the late result of ulcer of the stomach or duodenum. I confess my own leaning is toward the diagnosis of cancer. I am influenced to this partly by the rather vague history more closely resembling the dyspepsia not rarely associated with chronic alcoholism than the stomach symptoms of a gastric or duodenal ulcer. It seems to me that the history properly referable to an organic lesion of the stomach is rather short and to that extent in favor of cancer. I am interested in the difference of opinion between the medical and the surgical consultant, their positions being the reverse of those usually held. In this instance the physician is bloodthirsty while the surgeon is cautious. I think on the whole I should have agreed with the medical consultant on the ground that there was definite evidence of organic obstruction, that she was not likely to show definite improvement living in the somewhat haphazard way which has characterized her past, and that therefore no consistent improvement could be expected, even though she had a benign lesion. Clearly if one takes the view that this lesion is malignant delay might sacrifice the only opportunity for cure.

Her history after discharge shows that she did about what might be expected in regard to neglecting treatment. One could not of course have assumed that she would have run foul of a street car, and this of course should not be charged against her. However, her condition when she returned to the hospital three months later was clearly considerably less favorable than at her previous entry, and in light of hindsight valuable time had been lost.

The examination does not show anything important different from her previous admission except that the tenderness to the left of the midline in the upper abdomen appears to have increased.

Looked at as an operative risk she cannot be regarded as a good one. Her circulation is not in good shape. She may perhaps be regarded as a chronic alcoholic and if so be expected to stand operation poorly. She probably has some chronic bronchitis, a condition which is exceedingly likely to be flared up by operations in the upper abdomen, after which in any case lung complications are relatively frequent.

Considerable care should be taken in the selection of an anesthetic. I should myself prefer to begin the operation under local anesthesia and use either nitrous oxide or ethylene gas to carry on the portion requiring manipulation, finishing the operation with the remnant of the local anesthesia.

I think that the indication here will be to provide drainage for the stomach by gastroenterostomy. I do not think that a partial gastrectomy either for ulcer of the stomach or for cancer can be carried out with the patient in this condition.

I incline to the diagnosis of cancer as against chronic ulcer.

DR. CABOT'S PRE-OPERATIVE DIAGNOSIS

Carcinoma of the stomach.

PRE-OPERATIVE DIAGNOSIS

Ulcer of the stomach.

OPERATION

Gas-ether. Eight-inch midline upper abdominal incision. A very hard nodular growth with a deep ulcer was felt on the lesser curvature of the stomach close to the pylorus. This did not seem adherent posteriorly. The liver was grayish white, but was not nodular. The growth was resected with a good margin and posterior gastroenterostomy was done. A few glands of both the lesser and the greater curvature were involved. The gall-bladder was thin walled, contracted, and full of stones. It was not removed. Wound closed without drainage.

PATHOLOGICAL REPORT

The pylorus with the adjoining portions of the duodenum and stomach, measuring 7 cm. along the lesser curvature. Portions of both omenta are attached. On section there is a hard tumor 3 cm. in diameter which fills the pylorus and extends into the adjoining portions of the stomach. The mucous surface is superficially ulcerated and nodular. Its peripheral portions on the stomach side are not sharply marked off. On the duodenal side there is a sharp demarcation. There are liberal margins of sound tissue on all sides of the growth. In the lesser omentum there is a cluster of enlarged lymph nodes situated in close relation to the lesser curvature.

A microscopic examination of the growth shows an extensive invasion of the walls by irregular gland tubules composed of atypical epithelial cells with extensive reactionary fibrosis.

Four lymph nodes from the lesser omentum show in only one a metastatic focus situated in the peripheral sinus.

Adenocarcinoma with metastases in the regional lymph nodes.

H. F. Hartwell

Further Discussion

I quite appreciate the dilemma in which the surgeon found himself, namely, with a lesion which did not seem to him likely to be benefited by simple gastroenterostomy because it was not largely obstructive. On the other hand the probability of success following resection in a patient in this condition was, I think, small. I believe that I should have been satisfied with a gastroenterostomy taking all the circumstances into consideration. Her death was probably partly due to the development of a process in the lungs, but she was the sort of patient who would probably have died from some cause or other had her convalescence been more prolonged.

Necropsy should show arteriosclerosis, hypertrophy and dilatation of the heart, possibly arteriosclerotic nephritis, possibly some fibrosis of the liver, probably wet bases of the lungs or even definite bronchopneumonia.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the stomach.
Bronchopneumonia.
Operation, partial gastrostomy.
Cardiac failure.

DR. HUGH CABOT'S DIAGNOSIS

(Cancer of the stomach.)
Arteriosclerosis.
Hypertrophy and dilatation of the heart
Possibly arteriosclerotic nephritis.
Possibly some fibrosis of the liver.
Probably wet bases of the lungs or even definite bronchopneumonia.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

(Adenocarcinoma of the stomach.)

2. *Secondary or terminal lesions*

Arteriosclerosis.
Chronic adhesive pericarditis.
Hypertrophy and dilatation of the heart.
Bronchopneumonia.
Acute pleuritis, right.
Soft spleen.

3. *Historical landmarks*

Gastrostomy and gastroenterostomy.
Chronic pleuritis.

Dr. Richardson: The muscles were rather pale and soft.

About the lower third of the stomach was wanting. The lower border of the remaining portion presented an intact row of sutures closing it off. At a point above this an anastomosis was established between the first portion of the jejunum and the stomach. It was in good condition and admitted the passage of two fingers. The stomach otherwise was negative.

The upper end of the duodenum was sutured off and the part was in good condition.

In the right pleural cavity there was about 100 c.c. of thin cloudy fluid and fibrin. On the left side no fluid. There were a few old adhesions on the right, and on the left the lung was bound down by old adhesions. The trachea and bronchi contained considerable reddish frothy mucous fluid. The mucosa was negative.

The tissue of the right lung generally was spongy to a little leathery and showed considerable edema. In the region of the lower lobes there were scattered areas of bronchopneumonia, some just beneath the pleura, over which there was a thin fibrinous exudate. There were a few areas of bronchopneumonia in the upper lobe posteriorly. The tissue of the left lung generally was spongy, pale red—moderate edema.

The pericardial cavity was obliterated by old membranous fibrous adhesions. The heart weighed 400 grams, moderately enlarged. The organ was pulled over to the left by the old adhesions so that the right border was in a line with the middle of the sternum and the apex in the fifth interspace, axillary line. The myocardium was flabby and dirty pale brown red. Epicardial fat in large amount, and there was some fatty infiltration of the myocardium, best marked on the right. The valves were negative. The right side of the heart showed moderate dilatation. The coronaries were free and negative.

Aorta capacious. Ascending thoracic circumference 8½ cm., descending 6 cm., abdominal 6 cm. The intima of the ascending thoracic was quite smooth. The arch showed a slight amount of fibrous sclerosis and some fibrocalcareous areas, atheromatous in places. The descending thoracic showed a slight amount of fibrous sclerosis. The abdominal portion showed much fibrous sclerosis and several fibrocalcareous areas, atheromatous in places. The great branches were capacious and showed a slight to moderate amount of fibrous sclerosis. There were a few calcareous places in the iliacs.

The liver weighed 1135 grams, rather small. The surface was smooth, the tissue punky and pale. No metastases were made out.

The gall-bladder was closely applied to a mass of stones, ten of them. They were facetted, roughly pyramidal, and about 1 cm. in greatest diameter. They were grayish-brown and crushed under the thumb, showing crystalline material. The wall of the bladder showed no definite thickening and was closely applied to the stones. The bile ducts were free and negative.

The spleen weighed 64 grams, small. The surface was smooth and the tissue purplish red and mushy.

The head was not examined.

The case anatomically is one of bronchopneumonia with acute pleuritis in an arteriosclerotic with chronic adhesive pericarditis and hypertrophy and dilatation of the heart associated with the operation mentioned.

FURTHER DISCUSSION BY DR. CABOT

There is not much in the necropsy findings which can tempt us into further discussion, but I feel inclined to make reference to the old controversy of the probability of the development of cancer on ulcer. This doctrine has had a pretty wide vogue and has by some people been regarded as an indication for radical operation when it would not otherwise have been indicated. This case from its history might be regarded as of the ulcer-cancer type. The pathological report by Doctor Hartwell seems to me to make it quite clear that no such theory is tenable in this case. It is my own view that the probability of malignant degeneration of an ulcer of the stomach is not importantly greater than of the malignant degeneration of an ulcer of the leg. As we do not advise amputation for varicose ulcer in order to prevent cancerous degeneration, we should not, I think, be influenced in our choice of an operation for ulcer of the stomach by what seems to me a rather remote possibility.

CURRENT LITERATURE

ABSTRACTORS

Gerardo M. **Balboni**
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Laurence D. **Chapin**
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Myositis Ossificans in the Army Following Horseback Injuries to the Thigh

Bowen, A. (*Archives of Surgery*, Part 1, November, 1924).

Bowen reports two cases in detail and devotes the rest of his article to discussing prevalence of the conditions, its classification, etiology, pathology, diagnosis and treatment. He believes that the etiology is not absolutely determined; it is probable that both a diathesis and the exciting cause are necessary to its production. The principal problem in diagnosis is to rule out sarcoma and not treat this benign lesion as malignant. A good X-ray will usually enable the diagnosis to be correctly made. When in doubt, an exploratory incision for diagnosis is permissible. Conservative treatment without operation unless function is interfered with, and then only after all growth has ceased, is the rule.

[E. H. R.]

A CASE OF PRIMARY MULTIPLE ENDOTHELIOMA OF BONE

KOLODNY, A. (*Archives of Surgery*, Part 1, November, 1924).

This author presents an article based on the report of one case with extensive X-ray findings. It is of interest because of its rarity.

[E. H. R.]

BRODIE'S ABSCESS

HENDERSON, M. S., and SIMON, H. E. (*Archives of Surgery*, Part 1, November, 1924).

These authors present a study taken from the surgical clinic of the Mayo Hospital, and draw the following conclusions:

1. The symptoms of Brodie's abscess are characteristic, and may be explained on a pathologic basis.
2. It is a focus of infection situated in the spongy portion of the long bones near the epiphyseal line, arising either as a remnant of a former osteomyelitis, or of unexplained origin.
3. There is often a period of latency which may be quite long, during which the resistance of the body and the local wall of sclerosed bone at the abscess margin holds the process in abeyance.
4. It may burst into activity as a result of lowered resistance, due in some cases to trauma or acute infection.
5. The occasional complications are: (a) sinus formation with sequestrum, (b) extension into the diaphysis, (c) effect on general health, and (d) marked enlargement.
6. The condition is not tuberculous in origin.
7. Surgical treatment suited to the location in the bone, and to the type of the abscess, results in cure in the majority of cases. Recurrence may occasionally be met with after surgical treatment.

[E. H. R.]

SQUAMOUS-CELL TUMORS OF THE RENAL PELVIS

SCHOLL, A. J., and FOULDS, G. S. (*Annals of Surgery*, October, 1924).

These authors present an article regarding the history of five recent cases, and draw the following general conclusions:

"Squamous-cell tumors of the renal pelvis probably result, in most cases, from chronic irritation, are highly malignant, rapidly involve the renal parenchyma and neighboring tissues, and readily metastasize. This type of growth is at times found in association with renal calculi, which are rough, irregular, and of tremendous size. Owing to the lack of symptoms suggesting a malignant condition, these tumors are rarely seen when the growth is small or localized to the renal pelvis. The operative mortality is high, and the majority of patients who survive die shortly after the operation from local recurrences, or metastatic growths.

"Five cases of squamous-cell tumors of the renal pelvis have been observed at the Mayo Clinic between 1907 and 1922. Four of these cases were associated with renal stone; the calculi in three kidneys were extremely large and of the staghorn type. One patient died eight days after operation, three others died during the first four months. The fifth patient is alive without symptoms of recurrence six months after operation."

[E. H. R.]

LATE RESULTS OF SPLENECTOMY FOR TRAUMATIC RUPTURE OF THE SPLEEN

PFEIFFER, D. B., and SMYTH, C. M. (*Annals of Surgery*, October, 1924).

These authors find that:

1. Individuals splenectomized for traumatic rupture usually show a definite and persistent anemia which in our series did not fully recover under the ordinary conditions of life, and therefore requires prolonged observation and treatment.
2. That the spleen exercises an important function is evidenced by the hyperplasia of lymphoid tissue throughout the body, and of splenic tissue which remains after splenectomy.
3. Decreased bodily vigor and resistance, often noted clinically and experimentally, may depend on anemia, but in the present state of our knowledge it is impossible to exclude endocrine or metabolic disturbances as yet unknown.
4. There is no evidence of such adverse influence on health or longevity as to contraindicate splenectomy for traumatic rupture, which is ordinarily the operation of choice.

[E. H. R.]

AN EXPERIMENTAL CONSIDERATION OF THE INFECTION OF PERMANENT HYDRONEPHROSES

DAVID, V. C., and MCGILL, E. C. (*Annals of Surgery*, October, 1924).

These authors from experimental work conclude that closed hydronephroses in dogs may be infected through the blood stream up to three months after the onset of their development; that closed hydronephroses of more than three months' standing are no longer liable to infect through the blood stream; that closed hydronephroses are rarely, if ever, infected through the lymphatics leading from the bowel to the kidney; and that ascending ureteral lymphatic infection of hydronephroses is possible but improbable.

[E. H. R.]

PROTEIN BODY THERAPY

WEICHARDT (*Wien. klin. Woch.*, July 17-24, 1924) reports a series of clinical studies on the theoretical foundations of protein body therapy, whose object he regards as the activation of the organism, changing the reaction of cells in the sense of increasing their functional capacity.

[R. M. G.]

THE USE OF FREE TRANSPLANTS OF FASCIA AS LIVING SUTURES IN THE TREATMENT OF HERNIA

GALLIE, W. E., and LEMESURIER, A. B. (*Archives of Surgery*, Part 1, November, 1924).

These authors present a brief review of their experimental work with fascia lata in the cure of hernia. They describe their operative technic and present some very valuable drawings. They believe this method of using living tissue is the only adequate one against recurrence in many types of hernia, especially the recurrent types and large ventral herniae which have recurred after the most painstaking attempts of cure by the ordinary methods of suture.

[E. H. R.]

DIFFERENTIAL DIAGNOSIS OF ENLARGED HEART AND PERICARDITIS WITH EFFUSION

PERKINS, WINFIELD C., and LATTIN, BERTON (*American Journal of Roentgenology*, Vol. XII, No. II, p. 171).

Pericarditis may cause an enlargement of the roentgen shadow in two ways:

A. Pericardial effusion, especially of medium or large size.

B. Chronic adhesive pericarditis. Here the heart's action is impaired by numerous adhesions which tend to bind the heart to the pericardial sac, and compensatory hypertrophy of the heart muscle results. Hearts of this type show the largest size x-ray shadow of all. The most important x-ray signs are:

1. A widening of the shadow enveloping the great vessels.
2. Absence of pulsation of the left border of the heart as seen by fluoroscope. If there is vigorous pulsation of the left border and apex, there can be little fluid present.
3. Actual demonstration of the heart shadow within the pericardial shadow, the latter being distended by fluid.
4. The water-bottle shape of the heart when rayed in the vertical position and the change in shape of the x-ray shadow with change in posture. It is pointed out that this finding is of much less value than might be supposed because in most cases of large effusion orthopnoea is almost always present, making it difficult to obtain a plate in the recumbent position. Another point brought in the x-ray shadow of the pericardial effusion: the cardiohepatic angle is more acute rather than more obtuse.

[H. A. O.]

PARAFFINOMA OF THE VAS DEFERENS

WATSON, LEIGH F. (*Journal of the American Medical Association*, 1924, lxxxii, June 14, pp. 1935-1936).

The author reports an unusual complication following the paraffin injection of an inguinal hernia by a charlatan. The hernia promptly recurred and the cord and testicle on the side treated became swollen, painful and tender on pressure. At operation paraffin masses were removed from the internal oblique muscle, the conjoined tendon, and the vas deferens. The vas was occluded and it was necessary to resect it and anastamose the ends.

Because of the high percentage of recurrence following operations for "paraffin hernia" the regular Bassini operation was combined with the author's method of *lateral displacement of the cord*. With the cord displaced on to the internal oblique, one-half an inch to the inner side of the deep suture line, the overlapped fascial flaps were securely stitched to the deep suture line to reinforce the weak spots, the usual points of recurrence—the internal ring, the lower end of the incision over the pubic bone, and the line of deep sutures.

The serious accidents that sometimes follow paraffin injections of hernia are: Gangrene of the skin; injection of cord structures; wounding of intestine, appendix or bladder with the needle; injection into blood vessels, followed by pulmonary or cerebral embolism or sudden blindness from plugging of the artery of the retina, and occlusion of the iliac or femoral artery, with gangrene of the extremity, necessitating amputation.

PSEUDOMYXOMA PERITONEI

RIES, E. (*Surgery, Gynecology and Obstetrics*, November, 1924).

This author discusses this condition and draws the following conclusions:

1. Pseudomyxoma peritonei is a secondary condition due to the escape of gelatinous material from a primary tumor. This tumor arises in the ovaries or in the appendix or both or rarely in other organs.
2. The ovarian tumors are always pseudomucinous kystomata in their entirety or at least partially.
3. The appendices from which pseudomyxoma peritonei starts are always more or less cystic, often show diverticula, and may contain papillomatous tumors.
4. The operative treatment is successful in proportion to the completeness of removal of the original tumors and the pseudomyxoma peritonei.
5. Every case of pseudomyxoma peritonei, especially in the female, requires investigation of the appendix and its removal.
6. The chemistry of the gelatinous masses deserves further study with proper attention to the sources of the colloid material.
7. The coexistence of ovarian tumor with appendiceal cyst in pseudomyxoma peritonei is a frequent finding. There is at present no evidence of anything more than accidental coexistence of the two conditions.

[E. H. R.]

INFLAMMATORY CARCINOMA OF THE BREAST

LEE, B. J., and TANNENBAUM, N. E. (*Surgery, Gynecology and Obstetrics*, November, 1924).

These authors report on 28 cases from the Breast Clinic of the Memorial Hospital. The article is profusely illustrated and they make the following statements as a result of their observations:

1. Inflammatory carcinoma appears to be a distinct clinical phase of carcinoma of the breast.
2. Generally, this type has been unrecognized, being frequently mistaken for other diseases of the breast.
3. The inflammatory appearances are characteristic.
4. This variety of mammary cancer shows no constant pathological type.
5. The most striking pathological change is a wide invasion of dermal lymphatics by carcinoma.
6. Bacteriological and biochemical studies have failed to explain the inflammatory manifestations.
7. The cases do badly if treated surgically.
8. At present radiation offers the only hope of palliation, diminishing the patient's suffering, and giving a definite prolongation of life.

[E. H. R.]

THE TREATMENT OF BRAIN ABSCESS BY UNROOFING AND TEMPORARY HERNIATION OF ABSCESS CAVITY WITH THE AVOIDANCE OF USUAL DRAINAGE METHODS

KING, J. E. J. (*Surgery, Gynecology and Obstetrics*, November, 1924).

This author presents a very interesting article with excellent drawings on the technic which he has developed in the treatment of this condition. He also discusses the management of hernia cerebri in general. The article is of interest and value.

[E. H. R.]

A RARE CASE OF UNILATERAL KIDNEY, WITH FUSION OF BOTH POLES

LAROSE, VICTOR J. (*Journal of Urology*, August, 1924).

Review of the literature by the author failed to reveal report of a case of fused kidney such as is here described, where the kidneys were fused at both the upper and lower poles to form a ring-like mass, with the pelves and calices situated directly opposite to the normal relations.

[B. D. W.]

SYPHILIS OF THE EPIDIDYMIS

ROLNICK, H. C. (*Journal of Urology*, August, 1924).

Syphilis of the epididymis occurs more frequently than a review of the literature would indicate, in the belief of the author, who here reports the histories of four cases. Occurring more often as part of the secondary manifestations, involvement of the epididymis is as temporary as the secondary eruption, and as there are few symptoms of diagnostic importance it escapes attention of the patient and physician. The condition is usually secondary to or associated with syphilitic orchitis; primary syphilitic epididymitis without involvement of the testicles is rare, but does occur. In syphilis, the testicle is more commonly involved than the epididymis because of its more direct blood supply and also because of the particular affinity of the spirochete for that organ.

[B. D. W.]

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BIRTH CONTROL AGAIN BEFORE CONGRESS

A BILL has been introduced in the National House of Representatives (68th Congress, First Session. H. R. 6542) to allow the circulation of contraceptive knowledge and means, provided "such information or means shall be certified by not less than five graduate physicians lawfully engaged in the practice of medicine to be not injurious to life or health."

It seems probable, although difficult of proof, that already contraceptive measures are employed with more or less success in the great majority of American families. Those who do not resort to this measure fall into two groups —those who refrain from religious motives, and those who through carelessness or from a desire to let nothing interfere with sexual gratification make no effort to prevent the natural consequences of the act.

That the latter class is rapidly diminishing is proved by the relative scarcity of really large families; the high standard of living in this country and the desire to give to children the advantages which will advance them to a higher social plane than their parents, encourage the limitation of offspring. It is unlikely that ignorance of contraceptive measures *per se* is an important factor in the situation, for where there is the will to find out such methods, there is a way. Certain types of prevention are indeed a matter of common knowledge.

These measures frequently fail, but they undoubtedly serve to keep down the birth rate. Whether they ought or ought not be employed cannot properly be argued in these columns; that is a question for each couple to decide. The fact is that information about contraceptive measures is easily acquired, either from friends, parents or a doctor.

Inasmuch as this information is so widely spread and so extensively employed, it seems unfortunate that there are contained in the General Laws (Chap. 272, Sect. 20 & 21) two sections making it a crime punishable by fine or imprisonment for anyone to advertise or "sell, loan, give away or exhibit" any "drug, medicine, instrument or article whatever" for the prevention of conception. The law is practically a dead letter, for there is no difficulty in purchasing such drugs or articles.

Insofar as the bill recently introduced into Congress does away with this false situation, it is to be commended. Is there, however, a valid reason for removing the restrictions now placed by law upon the circulation of such knowledge, and are there dangers attendant upon the removal of such restrictions? In our opinion, the dangers would outweigh the benefits of such legislation.

The advantage that would be derived from placing before the people easily accessible information concerning the best contraceptive methods would be slight. There is admittedly no ideal method, and none that, while harmless, is absolutely certain. The methods now in use are seldom harmful, even if they do not always function. If a better method were to be discovered, knowledge of it would spread with the greatest rapidity. On the other hand, if the removal of restrictions were taken advantage of, it would be through the efforts of a few enthusiasts, who might stimulate an interest in the subject which would extend farther than was anticipated. Or it would be done by those who would exploit contraceptive drugs and apparatus for commercial purposes. Even the requirement of a certificate signed by five graduate physicians would not check this. The cheaper type of newspaper and periodical would carry advertisements setting forth extravagant claims, and keeping the topic constantly before the eyes of the younger generation.

We cannot see any reason for allowing such a display of information which can so much more fittingly be conveyed by word of mouth to those who desire it seriously enough to take the trouble to ask for it.

SOME REMARKS ON ENCEPHALITIS LETHARGICA AND ITS TREATMENT FROM THE VIEWPOINT OF ENGLISH PHYSICIANS

While a large number of medical observers have stated their conviction that encephalitis lethargica is a disease by itself, there are still many who disagree with this opinion and record their view that it is a nervous form or one of the nervous forms of influenza. Perhaps there is as much to be said for one side as for the other side of the argument but it should be borne in mind that the line of treatment decided upon is largely dependent upon the personal views held concerning the nature of the disease. As Dr. F. G. Crookshank in an article published in the *Franco-British Review*, November, 1924, has pointed out, the available facts regarding encephalitis are largely historical. And those who have "views" and who seek a rational method of treatment in accord therewith fall into two groups. In the first group are those who hold that the cases represent the incidence of a specific disease, a disease *sui generis* produced by infection with a specific germ that has a special affinity for the parts of the brain that it "attacks." Those who think thus are looking forward hopefully to the discovery of a specific antitoxin or vaccine that will prove of curative as well as immunizing value. In the second class are those who take the stand that the so-called cases of encephalitis lethargica are really clinically and epidemiologically manifestations of influenza especially affecting the central nervous system. This point of view, too, has received pathological support at the hands, among others, of Tarozzi and Rosenow. Crookshank holds, moreover, that this view of the relationship between what is termed influenza and what is termed epidemic encephalitis or encephalitis lethargica goes hand in hand with the twin theses: 1. That no clear line of demarcation other than the topographical can now be maintained between "epidemic encephalitis" and "epidemic poliomyelitis" and 2. That "epidemic poliomyelitis," if not every case or type of so-called "infantile paralysis," stands in a similar position with regard to influenza as encephalitis lethargica.

Brorström was the first, some fifteen years ago, to suggest that epidemic poliomyelitis is a nervous form of influenza which has received recently convincing support from investigation of the Tubingen epidemic of 1922, reported by Schall in the *Münch. med. Woch.*, 1923, 744,763. Consequently, the synthetic view is that encephalitis lethargica represents a nervous form of influenza which tends to occur in older patients and epidemic poliomyelitis, a like form of which is more frequent during youth. As Crookshank says there are no established pathological, clinical, bacteriological or epidemiological facts inconsistent with this conception, which is otherwise supported by a vast mass of detailed observation. In addition, it affords a basis for a rational plan of treatment very successful in practice.

In considering treatment attention may be drawn to a point which seems to be of great importance. The Greek physician and the great physicians of the Renaissance conceived the notion that catarrhs were the expression of a distillation of humor from the brain and spinal cord. So long as there was expression of this distillation by flux from a mucous membrane, by sweat or diuresis, all was well. But when the distillation was repressed nervous troubles arose, indeed the whole gamut of psychoses which are features of encephalitis lethargica and poliomyelitis. And arguing from these premises, they treated accordingly and it may be said with some success. In recent years it has been found that nervous complications in influenza are less to be dreaded when there is sweating than when the skin is hot and dry. Crookshank concludes that it seems as if no matter what the particular virus may be, there is in certain disorders a sort of balance between the vaso-motor conditions of the central nervous system on the one hand and the skin and mucous membrane and viscera on the other. Attention is directed to the point that those who adopt what may be called the influenzal conception of encephalitis have some basis, both in reason and experience, on which to found their treatment: some standard by which test can be made. So far as treatment is concerned, it is safe and sound practice whenever encephalitis lethargica suggests itself to the physician that the patient be ordered to bed and kept there as long as considered necessary. Careful cleansing of the mouth and nose should be insisted upon at once, as these organs are reservoirs of the "unknown virus." A free evacuation of the bowels is essential. Next proper action of the skin and kidneys must be secured. Warmth in bed, a warm bath and an old-fashioned diuretic mixture should not be omitted. Little food is necessary and as milk is heavy and septic and encumbers the alimentary canal, it should not be given. Since the outbreak of 1918, Crookshank has almost invariably ordered the addition to these measures of urotropin, salicylate of soda, and benzoate of ammonia to the diuretic mixture. These drugs are helpful; they are antispetic and with reason one or the other may be said to reach the cerebro-spinal fluid, the urine and the bronchial mucosa. Of course if there be marked lethargy more active steps must be taken and then if at the end of several days no obvious signs of improvement are shown Crookshank recommends the old-fashioned exhibition of mercury. This remedy in the form of grey powder and inunctions has given the most satisfactory results. Lumbar puncture when necessary, but only when absolutely necessary, for diagnostic or therapeutic purposes and then to be employed with great care. In special cases special drugs are indicated but according to the experience of Crookshank and his hospital colleagues treatment based on the lines sketched above has been followed by a freedom from case mortality and from sequelae that does not appear to be secured by those who treat their cases less actively and more "expectantly." The main interest of Dr. Crookshank's able article lies in his views and of those of a like opinion who regard both encephalitis lethargica (that is, epidemic encephalitis and not cases which are nowadays described as such) and epidemic poliomyelitis as nervous manifestations of influenza and not as specific diseases, the former as a rule attacking older persons than the latter. Also the theory of treatment founded on the belief that sweating is indicated to prevent or relieve the nervous symptoms, combined with diuretic, excretory, eliminant and antiseptic therapeutic measures, is suggestive. When it is stated further that such measures have been attended with excellent results, it seems to afford evidence that treatment founded upon the diagnosis that encephalitis lethargica and epidemic poliomyelitis are not separate disease entities, but are related to influenza, is in the main correct.

THE ESSEX SOUTH MEDICAL SOCIETY

Attention is called to the official nature of the meeting to be held July 14 at the Boston City Club. The standing of Professor Winslow warrants a large attendance and the subject of his address is of commanding importance.

MISCELLANY

THE INTERNATIONAL ASSOCIATION OF MEDICAL MUSEUMS

Sir William Osler Memorial

The memorial volume now being published under the auspices of the International Association of Medical Museums (representing its Bulletin No. IX) comprises a large series of personal and biographical articles reminiscent of the life and activities of the late Sir William Osler during the different periods of his career, written by intimate friends and associates. This was primarily intended as a contribution, from first-hand evidence, to our knowledge of those early cultural influences and biological and pathological researches which militated so largely to his development as a leading teacher and master of Clinical Medicine but it has been extended, through numerous valuable contributions received, to cover all phases of his many-sided life. The scope and plan of the work are outlined in charming introductory articles by Sir Clifford Allbutt and Professor William Welch, and it is completed by a classified Bibliography of Sir William Osler's publications (based on the chronological bibliography by Miss M. W. Blogg), and by a bibliography of "Writings About Osler."

The volume will be handsomely bound in cloth and contains over sixty illustrations, both photogravure engravings and half-tone prints. The edition is being privately issued by subscriptions to the volume paid in advance, and with the aid of a publication fund which was inaugurated in January, 1921, by generous initial contributions from the National Research Council of Washington, the late Sir Edmund Osler and the late Hon. Mr. Justice Featherstone Osler (brothers of Sir William Osler), Mrs. K. S. Reford, of Montreal, and Mr. J. J. Carty, of New York. The price of the bound copy is ten dollars ($10.00), and subscriptions to it, or contributions of a larger amount to the publication fund, are needed and invited.

Cheques should be made payable to the *International Association of Medical Museums* (*Osler Memorial Volume*) and may be addressed to Dr. Maude E. Abbott, Permanent Secretary, International Association of Medical Museums, 1300 Spruce St., Philadelphia, Pa.

Following is a list of the contributors to the volume to date:

Frontispiece: From a photograph presented by Lady Osler for publication in this volume.

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Classified Bibliography. Edited by Fielding H. Garrison and Henry W. Cattell.

Bibliography of "Writings About Osler."

THE PRIZE FOR CANCER STUDY

The Commission for the distribution of the Prize for Cancer study founded by Doctor Sofie A. Nordhog-Jung, in agreement with the Foundress, has resolved to distribute the Prize from now on only every two years to the double amount of the sum allotted heretofore, that is One Thousand ($1000) Dollars. The next prize will reach distribution in 1926.

THE DUCROS BILL

At the last session of the Louisiana Legislature there was passed the Ducros Bill, a law requiring a physical examination of the male before marriage, and a certificate that the applicant is free from venereal disease. In regard to this Bill, the medical profession, we are sure, agrees that the Legislature has enacted a law which will go far towards making marriage a less hazardous affair, and which will ensure better health for the progeny.

THE WORLD'S CHILDREN

INVESTIGATION OF SCARLET FEVER

Highest susceptibility to scarlet fever exists from the ages of one to two years, according to the results of tests made with 7,700 normal persons of different ages. Results of these tests are summarized by Dr. Abraham Zingher in the *American Journal of Public Health*. Nearly three-fourths of the children between one and two years of age were susceptible, but only 14.4 per cent of those from 20 years up. Dr. Zingher reports that investigations are being made to discover the results of immunization against scarlet fever and diphtheria in the case of 2,000 children under 6 years of age. Records of these children, who have been immunized, are being kept for comparison with lists of scarlet fever and diphtheria cases developing during the coming year.—*Children's Bureau, Washington.*

NINETEEN TWENTY-FOUR MAY BE A RECORD HEALTH YEAR

As 1924 draws to a close, the following are the outstanding items in the year's health record. They are based on the cumulative deathrates of more than 15,000,000 Industrial policyholders of the Metropolitan Life Insurance Company, calculated up to November 15th.

The general deathrate for 1924 will probably be the lowest in the history of this important cross-section of the population of the United States and Canada.

The following facts are apparent concerning the mortality record for diseases of major public health interest. No doubt now remains that the year will register a new minimum deathrate for tuberculosis and for typhoid fever. The record for the principal epidemic diseases of childhood is highly satisfactory. The diphtheria deathrate is lower than ever before, and deaths from measles are fewer than last year. The whooping cough rate shows no increase despite the larger exposure to risk in 1924 of infant lives. The scarlet fever rate is a trifle higher than for last year, but is, nevertheless, below the average for the last decade. The mortality from influenza is less than half that of last year; a slight rise in the rate for diarrheal diseases is chargeable entirely to increased coverage of infant lives; the same is true of pneumonia.—*Statistical Bulletin Metropolitan Life Insurance Co.*

HUMIDIFY THE AIR IN HEATED DWELLINGS

Dry air is injurious to health and predisposes to sickness.

Air in artificially heated dwellings is deficient in moisture which is necessary for health and comfort.

Air should contain 50% of moisture but our overheated dwellings contain but from 25 to 30%.

When outdoor air is heated so as to maintain an even temperature of 70° but without the addition of watery vapor, its capacity for absorbing moisture is very much increased and will extract moisture from surrounding objects; furniture, woodwork and books crack and come apart, living plants wither and die. On humans it extracts moisture from the skin, mucous membrane and respiratory tract causing dryness of the skin, irritation of the air passages from loss of moisture by the secretions. It lessens resistance and working efficiency, causes general disturbance, lassitude, sleepliness, cardiac and nervous irritability.

Air deficient in moisture predisposes to pneumonia and bronchial affections and is a common cause of catarrh by causing the minute glands which moisten the air passages to secrete to excess to compensate for the loss by evaporation which continued becomes chronic and constitutes catarrh.

Children are very susceptible to dry air, become easily fatigued, apt to have headache and be listless, which interferes with school work.

Dry air is a carrier of dust, particularly when the moisture is less than 30%. Dust is a menace from its irritating properties, from being a carrier of germs, and from the irritation set up making the parts receptive to disease-carrying germs. Dry air, therefore, is detrimental to health from its general effect on the system, from its local effect on the tissues, and from its dust-carrying properties.

The condition is remedied by the evaporation of water in artificially heated dwellings. The water containers in furnaces and stoves are generally too small and apt to be neglected. The more practical way is to evaporate water in shallow pans placed adjacent to radiators and heaters and these, when attended to and kept filled, accomplish excellent results. Air properly humidified is more profitable and an economic saving in coal. It costs as much to heat a room from 58 to 72 as it does to raise the temperature to 58 on an average cold day.

Humidify the air and have better health, prevent sickness and save coal.—*Buffalo Sanitary Bulletin.*

IMPORTANT

A SURVEY of 55,000 children in the 150 elementary schools of Los Angeles, California, conducted by Dr. E. C. Beach, an eminent dietetic specialist, showed that the children who drank milk liberally completed the eight grades of school work on an average of two years younger than those who drank little or no milk. It further showed that 20% of the children who were non-milk drinkers were retarded approximately two years in their school work. As it averaged $75 of the public money to keep a child in school a year, it was evident that an enormous saving to the taxpayers could be made if more attention were paid to the serving of milk in the public schools. While a similar survey in Brookline would undoubtedly be more favorable, nevertheless those who do not drink milk regularly are sufficient in number to represent a large waste of public funds. All authorities agree that milk is the most necessary food for the growing child and one of the most important for adults. Thus the greater use of milk would materially assist in the educational, financial, and physical advancement of the community.

WILLARD E. WARD,
Agent, Milk and Food Inspection.

APPENDICITIS IN MENTAL HOSPITALS

IN a letter of a medical man to ***Truth*** it is stated that appendicitis is rarely met with in mental hospitals.

"This is not evident," he writes, "in the report of the Board of Control, where appendicitis is included with other gastro-intestinal diseases, but the fact is well known to all who work in mental hospitals. In a mental hospital containing one thousand patients there has been no case of appendicitis in the last two years, although in the same period there have been several among the staff."

CRITICISM

What inference are we to draw from the striking difference in the incidence of appendicitis among the insane inmates of this hospital and its staff? Are we to conclude that the mere fact of beng insane diminishes the tendency to appendicitis? This seems highly improbable. I suggest that, if during these two years the staff of this hospital had lived under exactly the same conditions as the inmates, all of them would probably have been exempt from appendicitis, and that, on the other hand, if the inmates had lived under the same conditions as the staff, there would have been several cases of this disease among them.

People who live a routine life on a simple diet rarely get appendicitis. It is no doubt essentially a dietetic disease. It would be interesting to compare the incidence of appendicitis in public with that in private mental hospitals.—*The Medical Press and Circular.*

THE SPLEEN INDEX IN MALARIA

In making a survey it is necessary to have some reliable measure of the amount of malaria existing. The experience of the past year has shown that in the United States, as well as in the tropics, the splenic index of children is probably the simplest and best although no single index is of value in all localities. Blood indices, clinical histories, and physicians' morbidity and mortality reports are all needed.

"In Lee County, Georgia, where a laboratory has been established for the study of certain field problems in malaria control, investigations were conducted by Dr. S. T. Darling during 1923 to determine the reliability of the spleen index as a measure of malaria. Although spleen examination is extensively employed in malaria diagnosis in the Orient it has not yet been generally adopted in the United States, and little has been done to ascertain the limits of its usefulness. It is a method of diagnosis requiring much less time than the taking of clinical histories or the examination of blood specimens. It is of particular value in work among children and among ignorant and dependent peoples particularly those speaking a foreign language or those whose testimony as to their symptoms is unreliable because of their tendency to make such answers as they think their interrogator wishes or, through fear or timidity, to deny all clinical symptoms.

"A comparison of the spleen rates, plasmodium rates, and malaria history index of 571 persons examined on thirty plantations in Lee County showed that the spleen index furnished a more sensitive measure of malaria than the blood index. These persons, whose malaria history index was 37.4, give a spleen index of 40.8 as compared with a plasmodium index of only 25.2.

"Spleen examination will detect cases of malaria acquired the previous season, which may act as carriers of the infection. It is these carriers that infect Anopheles mosquitoes in the spring and summer and give rise to epidemics or seasonal endemicity."—*Information Service of the Rockefeller Foundation.*

AVOIDING A COMMON COLD

A RECENT study of colds among babies brought to Infant Welfare Stations in Chicago showed a rather high incidence of colds in the families of babies having colds. This was especially true where the mothers had colds. In fact exposure to mothers' colds was stated to have occurred in nearly half as many babies without colds as of those with colds.

It is clear that outside influences particularly those involving chilling of the body may serve to induce a cold. Even in spite of our rather uncertain knowledge as to the cause of common colds we are able to point out methods of prevention which if followed should result in materially lessening incidence of colds.

Perhaps the most important method of prevention is to guard against too sudden strains on the heat regulating mechanism of the body. People who live in overheated houses are easily chilled upon going out in the wintertime. The temperature of dwelling houses and offices should be kept at not over 70 degrees Fahrenheit.

The other method of preventing colds consists in measures for preventing the spread of infection. While the specific infective agent is not known, the evidence available appears to warrant taking precautions based on the theory of the infectious nature of colds. This would simply mean the avoiding of close contact with persons who have colds.

One of the most important methods of avoiding such infections is to keep far enough away from infected persons so as to be out of range of "droplets" thrown out by coughing, sneezing, or talking.—*Connecticut State Department of Health Weekly Health Bulletin.*

MEDICAL MEN AND SECRET REMEDIES

THERE used to be an honorable tradition in the medical profession that it was discreditable to employ or to recommend secret remedies. A physician, as a man of education and scientific knowledge, was expected to know the nature of his remedies, and to understand at least something of their physiological action. So strongly was this view held that some of the colleges prohibited, and still prohibit, their members from any association with secret remedies. For ourselves, we think this attitude of mind correct, and we regret that there are many evidences that it is no longer general in the profession. A few weeks ago a pamphlet was issued "to the medical profession with the compliments of" several more or less important public personages. It is described as being "evidence" concerning a certain secret, but much-boomed, treatment for tuberculosis, and it contains, intermingled with letters from members of Parliament and other such authorities, extracts from articles and letters by various medical men dealing in a laudatory way with the treatment in question. The information given was not always very lucid, as, for example, the following sentence, which we take almost at random from one of these reports:—"By the use of different processes he isolates different types of toxins of different chemical constituents and of profoundly varying degress of virulence, as demonstrated by him in experiments both in vivo and in vitro." But the important thing is that these medical men are apparently men of repute, holding respectable positions in the profession. Some of them are Fellows of Colleges, one a Fellow of the Royal College of Physicians of London. We cannot but think that the old avoidance of secret remedies was sounder morally and scientifically than the modern patronage of the unknown.—*The Medical Press and Circular.*

MORTALITY FROM DIABETES MELLITUS: 1923

THE Department of Commerce announces that there were 17,357 deaths from diabetes mellitus in 1923 in the death registration area, which comprised about 88 per cent. of the total population of the United States. The death rate in 1923 was 17.9 per 100,000 population as compared with 18.4 in 1922.

Of the 34 States which show adjusted rates for 1923 and 1922, 10 show higher rates in 1923 New York had the highest adjusted rate (24 per 100,000 population) in 1923, and Mississippi had the lowest (7.2).

Deaths and death rates from diabetes mellitus in the New England States:

Area	Number of Deaths		Death rate per 100,000 population			
			Adjusted		Crude	
	1923	1922	1923	1922	1923	1922
Connecticut	336	327	12.5	21.3	22.8	22.6
Maine	175	195	16.5	18.6	22.5	25.3
Massachusetts	861	954	18.8	21.0	21.4	24.0
New Hampshire	124	139	20.4	22.9	27.7	31.1
Rhode Island	149	144	21.7	21.2	23.8	23.2
Vermont	78	95	16.8	20.5	22.1	27.0

DEPARTMENT OF COMMERCE,
Washington.

MEMORIAL ROOMS IN THE HARVARD MEDICAL SCHOOL DORMITORY

THIS was broadcasted over the Radio by STATION WEEI, Edison Electric Illuminating Co., Boston, on Tuesday, December 23, at 7.45 P. M., by Dr. Elliott P. Joslin:

On December 10 an appeal appeared in the Boston papers for the balance of the $1,000,000 needed for the Harvard Medical Dormitories, for the use of students of the Harvard Medical School. These young Medical School students are studying for a career which stands for the greatest in all careers. It means intense mental effort, great nervous strain, great physical endurance. In his life and letters, Robert Bacon tells how his heart was touched when he saw bands of young American men hurrying, full of enthusiasm, even laughing, to the battlefields of France and he realized, as they did not, what they would have to face.

So it is with these young medical students. Most of them are now unmindful of the personal sacrifices their calling will mean when they have entered upon it. Only those closely in touch, as are members of a doctor's family or patients, half realize what a modern doctor's life is. That is why it is so important that these young men should enter it fully equipped for its endurance.

These boys need just three things besides their work—good food, clean rooms and opportunity for exercise, such as tennis, squash or hockey. At present these necessities are lacking. These students sleep all over Boston and Brookline, often in cheerless quarters, and eat at restaurants—anywhere. No wonder boys are forced to leave Medical School with broken digestive or nervous systems.

A Dormitory might be built as a business project if high rentals were charged, but such charges would defeat the main purpose of this Dormitory—to afford good rooms to students of small means who need them most. A large part of the cost must therefore be defrayed by gifts, small and large, from whomever would be a friend to students of medicine.

Thirteen hundred and ninety graduates of the Harvard Medical School have already subscribed $95,000 and the total subscribed to date toward the Dormitory is $264,000. Approximately $500,000 more is needed and must be raised.

In this Dormitory certain rooms have already been given in memory of physicians who have died. There are many rooms which might so be donated, and this method of giving should make a special appeal to those of you listening in who would like to do honor to the memory of a physician. Some of the men for whom rooms have already been given entered the profession fully equipped, carried on their work with ever greater success and finally ended their careers with the realization of what they had given to the world. Some, when only at the opening of their careers, caught disease and died, or lived for years lookers-on at the success of their classmates—theirs was the harder fate. Both deserve honor.

Since the World War, Armistice Day has been a time at which the living paid a tribute to the men who died in battle and to the unknown soldiers. It is just as fitting that Memorial Rooms should perpetuate the names of that army of doctors who, whether young or old, have died after offering their lives for the good of humanity, whether as family doctors or specialists, or in the laboratory or in the Great War. In whatever way they worked they deserve these Memorial Rooms. There will probably never again be such an opportunity to testify our gratitude to this body of men.

Some of the Memorial Rooms have been given now, but only a few. Each represents $2,500. Some of them were given by members of the committee who raised the money for their fathers. Among those which are finished some stand as memorials given by the family, or the family and friends, of the doctors. One of these rooms was given by eighty-four friends and patients of a physician who came to the Medical School a comparative stranger and before he was an old man had become so beloved as a friend and so successful as a physician that those who contributed considered it a privilege to help to raise this Memorial Room. His family did not know of it until it was completed, and no words can describe their joy that a memorial connected with the Harvard Medical School should stand as a perpetual monument to one who never thought of himself or in the least realized what others thought of him.

Complete details, with plans of buildings, will be sent you if you will write to Dr. Francis M. Rackemann, 263 Beacon Street, Boston, Massachusetts. Any one wishing to give a sum toward a Memorial Room without any special doctor in mind may specify that his check shall be used for that purpose. Checks should be made out to the order of Charles Francis Adams, Treasurer of Harvard University. If you wish for information you can write to Dr. Rackemann. Checks can be given to any one of the following Committee:

Dr. J. Dellinger Barney, Dr. John W. Bartol, Dr. John M. Birnie, Dr. Walter P. Bowers, Dr. John Lewis Bremer, Dr. George Emerson Brewer, Dr. Thomas J. Burrage, Dr. David Cheever, Dr. Henry A. Christian, Dr. Harvey Cushing, Dr. Elliott C. Cutler, Dr. George P. Denny, Dr. Marshall Fabyan, Dr. Michael F. Fallon, Dr. Maurice Fremont-Smith, Dr. Channing Frothingham, Dr. Homer Gage, Dr. James L. Gamble, Dr. Joseph Garland, Dr. Alan Gregg, Dr. Daniel F. Jones, Dr. Elliott P. Joslin, Dr. Roger I. Lee, Dr. Howard Lilienthal, Dr. Edwin A. Locke, Dr. James H. Means, Dr. W. Richard Ohler, Dr. Francis W. Peabody, Dr. William C. Quinby, Dr. Francis M. Rackemann, Dr. William L. Richardson, Dr. Milton J. Rosenau, Dr. George G. Sears, Dr. Frederick C. Shattuck, Dr. James S. Stone, Dr. John B. Swift, Dr. Philemon E. Truesdale, Dr. J. Collins Warren, Dr. James C. White, Dr. Nathaniel K. Wood, Dr. Samuel B. Woodward, Dr. Alfred Worcester.

FATALITIES FROM ACCIDENTS: 1923

Washington, D. C., December 15, 1924.—The Department of Commerce announces that in 1923 practically one-fifth of all fatalities from accidents were the result of automobile accidents. Approximately twice as many deaths from automobile accidents occurred in urban as in rural districts; a difference doubtless due, in part, to the greater number of hospitals in urban districts.

Of the 38 states, only three—Kentucky, Mississippi, and Wyoming—had higher mortality rates from railroad accidents than from automobile accidents. Wyoming had the highest mortality rate from railroad accidents (34 per 100,000 population), and Massachusetts the lowest (3.5).

Wyoming also had the highest mortality rate from all kinds of accidents (195.4 per 100,000 population), and Mississippi the lowest (57).

California had the highest mortality rate from automobile accidents (32.6 per 100,000 population), and also from street car accidents (4.7), while Mississippi had the lowest rate from automobile accidents (4.4 per 100,000 population), and Wyoming had no deaths from street cars.

Of the 66 cities of 100,000 population and over, for which rates are shown, Camden had the highest mortality rate from automobile accidents (16.4), and also from all accidents (144.5).

On the other hand, the lowest rate from automobile accidents (6.2) was for New Bedford, and the lowest rate from all accidents (47.3) was for Fort Worth, while Duluth, Jacksonville, San Antonio and Yonkers had no deaths from street car accidents, and New Bedford none from railroad accidents.

The deaths from accidents in the New England States, and New England cities of 15,000 population, in 1923 were as follows:

	Estimated population July 1, 1923	Accidental deaths in 1923 from					
		Automobiles	Railroads	Street cars	Other vehicles	All other	Total
Connecticut	1,476,483	249	61	35	29	707	1081
Cities	1,117,699	208	45	31	24	563	871
Rural districts	358,784	41	16	4	5	144	210
Maine	777,259	91	33	12	19	426	581
Cities	225,748	36	14	9	5	148	212
Rural districts	551,511	55	19	3	14	278	369
Massachusetts	4,027,545	611	140	68	74	1935	2828
Cities	3,300,344	507	94	63	60	1617	2341
Rural districts	727,201	104	46	5	14	318	487
New Hampshire	447,592	59	25	2	7	248	341
Cities	200,809	33	14	2	6	115	170
Rural districts	246,783	26	11	—	1	133	171
Rhode Island	626,672	97	25	27	3	275	427
Cities	521,243	83	22	24	2	238	369
Rural districts	105,429	14	3	3	1	37	58
Vermont	352,428	46	17	1	6	201	271
Cities	48,822	15	—	1	—	32	48
Rural districts	303,606	31	17	—	6	169	223

REMOVALS

Dr. Marion Nute has moved from Dorchester to Brookline, office Boston, 483 Beacon St.

Dr. J. H. Swartz of Dorchester now has an office at 536 Commonwealth Ave., Boston.

Dr. H. Allan Novack of Brookline now has a Boston office at 483 Beacon St.

Dr. Florence F. Rice has moved from 140 to 77 Thorndike St., Brookline.

Dr. Nathan L. Burnett has moved to 20 Austin St., Cambridge.

Dr. William F. Cotting of Cambridge now has a Boston office at 395 Commonwealth Ave.

Dr. William K. S. Thomas of Cambridge now has a Boston office at 510 Commonwealth Ave.

Dr. Joseph M. Wadden has moved from 69 to 159 Concord Ave., Cambridge.

Dr. William D. Reid of Newton now has a Boston office at 491 Commonwealth Ave.

Dr. Walter B. Lancaster has moved from Newton Center to Brookline. His office is Boston, 520 Commonwealth Ave.

Dr. Armin Klein has moved from Chelsea (Suffolk) to Brookline (Norfolk). His office is at 483 Beacon St., Boston, as before.

Dr. Theodore C. Beebe's present address is Paris, France, 21 Avenue George V.

Dr. Charles A. Bates has moved from Ashburnham to Northfield, N. J. His office is in Pleasantville, that state.

Dr. Benjamin S. Sharp, formerly of Boston, is now instructor in otolaryngology in the State University of Iowa Medical College, at Iowa City, Iowa.

RECENT DEATHS

KELEHER.—Dr. Francis Joseph Keleher died at his home in Somerville, January 1, 1925, at the age of 58.

Dr. Keleher was a graduate of Boston College, class of 1886; Harvard Medical School, class of 1890, and Boston University Law School, class of 1895, being admitted to the bar in 1897.

Previous to his final illness, a year ago, he was connected with the Bureau of Vital Statistics for the city of Boston. From 1892 to 1908 he was a Fellow of the Massachusetts Medical Society, then having a residence in Dorchester. For fourteen years he was lecturer on medical jurisprudence at Tufts College Medical School.

Dr. Keleher is survived by his wife, one son, the Rev. Francis X. Keleher of the Blessed Sacrament Church, Jamaica Plain; another son and three daughters.

WOOD.—Following a period of illness at Phillips House, Massachusetts General Hospital, Dr. Thomas Weston Wood, Jr., died there at the age of 63 years. Dr. Wood was a Back Bay dentist with offices at 160 Newbury Street, where he had been for a number of years. He was a graduate of Brown University in the class of '84, and subsequently took a course at the Harvard Dental School, from which he was graduated in the class of '87. From Brown he received his A.M. and A.B., and he was a member of the Phi Beta Kappa. He was a talented musician, specializing in the study of the violin.

Dr. Wood was born in Newport, R. I., the son of Thomas W. Wood. His only survivor is his wife, who was Elizabeth Adams of Roxbury.

YOOSUF.—Dr. Abraham Kevork Yoosuf, a Fellow of the Massachusetts Medical Society, died in his office in Worcester, of heart disease, December 26, 1924, aged 58.

He was born in Harpoot, Asiatic Turkey, in 1866, a son of a silk manufacturer. He came to New York in 1894, where he earned money for a medical education. He was graduated from the Baltimore Medical College in 1895 and began practice in Worcester in 1905, when he joined the State medical society.

During the Balkan War he was chief operating surgeon in a hospital in Constantinople, and was later decorated by the Sultan of Turkey for meritorious service. During the World War Dr. Yoosuf served as a major in the United States Army Medical Corps. After the close of the war Dr. Yoosuf spent two years in Paris working in the cause of Assyrians. Latterly he had been active in raising money for an Assyrian Church in Worcester. He was a member of the American Medical Association.

OBITUARY

WALTER E. FERNALD, M. D.

Dr. Walter E. Fernald after a brief illness, which came on him in the midst of full activity, died at Waverley on Nov. 26, 1924; thus ended a life of rare distinction and noble endeavor.

WALTER E. FERNALD, M.D.

Dr. Fernald was born at Kittery, Maine, in 1859; brought up on a farm, he received his preliminary education at New Hampton (N. H.), taught in a country school, was apprentice to a Portland physician, graduated from the Medical School of Maine (Bowdoin) in 1881. From 1882 to 1887 he gained experience in mental disorders at the Mendota State Hospital, Wisconsin. In 1887 he was appointed Superintendent of the Massachusetts School for the Feeble-Minded, then situated in South Boston; he supervised with the greatest skill the construction of a new institution at Waverley and by 1893 completed the transfer of his 400 charges to their new school.

At Waverley Dr. Fernald gradually built up an organization for the training of the feeble-minded, which became a model and attracted visitors from all over the world. He brought to bear on the problems of the feeble-minded a profound sympathy, a rare tact, and a religious devotion and worked with untiring industry at the solution of the scientific problems they presented.

It is difficult to do justice to the varied aspects of his activity. He supervised with conscientious care the study and training of his handicapped wards, outlining to the last detail their daily program. He grouped around him able teachers and inspired them with his devotion. He instructed physicians, medical students, psychologsts, social workers, nurses. He lectured at Tufts Medical School, and at Harvard University, from which in 1918 he received the honorary degree of M. A., he gave courses both in the Graduate School of Education and in the Graduate Courses in Medicine. He contributed to medical literature the results of his special researches and of his maturing thought. He inspired wise legislation in the interests of the mentally handicapped. He realized that, without the coöperation of an enlightened public, scientific accomplishment may remain sterile, and he gave much time and energy to the promotion of this coöperation.

During his life-time notable contributions were made from various directions to the study of the mentally defective and to them all he showed a receptive and enquiring attitude. The introduction of the Binet-Simon tests gave a method of precision which he utilized with wise discrimination, for his own observation and keen intuition had given him a body of experience which enabled him to place new data in their true perspective. His main interest was in broad clinical observation, but the anatomical basis of the phenomena he observed was a subject of earnest investigation, which he carried on along with Dr. E. E. Southard and other collaborators. The Waverley Researches on the Pathology of the Feeble-Minded (1st Series, May 1918; 2nd Series, Dec. 1921; 3rd Series, in preparation) represent the high-water mark of research on the pathological anatomy of feeblemindedness in this country. His receptive mind made him eagerly scan the promises held out by the new discipline of endocrinology, and willingly advocate a thorough trial of the new methods.

He himself has reviewed the development of thought in relation to the topic of feeble-mindedness in his Presidential Address to the American Association for the Study of the Feeble Minded, May 1924, in which he looks back to the situation in June 1893, when he held the same honourable office and delivered his first Presidential Address to the Association. The first two decades of that period had been fertile in investigation, and had ended in pessimism; "intelligence" tests had focused attention in a precise way on an irremediable defect, studies of the heredity of "hovel" defectives had demonstrated the source from which they sprang and the atmosphere in which they lived, analysis of gaols and penitentiaries seemed to show the end of their careers. The feeble-minded were considered as individuals not only of fixed intellectual level, but of criminal tendencies and with a deep-seated hereditary taint, they were useless and a danger, to be segregated and sterilized. This conception, so intolerable to his warm human sympathies, Dr. Fernald called the "Legend of the Feeble-Minded"; he showed that it was based on the study of the troublesome group of defectives, the only group studied during these earlier years, and he never wearied of calling attention to the still larger group of the mentally handicapped, often of amiable disposition, found sporadically in homes of every type, receptive of a training suited to their needs, able under kindly supervision to take a useful part in the economic life of the community. He placed the dark side of the picture in correct perspective, redressed the faulty balance, emphasized the assets as well as the liabilities of the mentally handicapped, and instead of dwelling entirely on the protection of the community from the liabilities due to this group he called attention to the equally important need of justice being done to these handicapped ones. In order that they may be an asset and not a liability, early diagnosis, appropriate training, kindly social supervision are imperative and the organization of the requisite provisions for these three activities devolves on the state and on the community at large. These views of Dr. Fernald came to full fruition when in 1919 the Massachusetts Legislature passed a law for the state-wide examination of mentally retarded children. When this law was passed Dr. Fernald devoted himself to its successful application; he did everything possible so that the contemplated survey should proceed with the maximum smoothness and efficiency; he gave a series of courses to the physicians, on whom the actual examinations devolved, and it was in the middle of such a course that he was stricken with his fatal illness. No one factor contributed so much to give the public confidence in the wisdom of the measure as the fact that it was personally sponsored by Dr. Fernald.

The importance which he attributed to the education of the community in regard to problems of mental health in general was shown by his active participation in state and national organizations. He was Vice-President of the Massachusetts Society for Mental Hygiene, of which, since its incorporation in 1912, he had been the constant inspiration. As Director of the National Committee for Mental Hygiene and Chairman of its Committee on Mental Deficiency, he helped to promote those systematic surveys of the needs of the mentally handicapped which led in many states to extensive constructive measures. He was President of the Boston School for Occupational Therapy in virtue of his consistent advocacy of the gospel of work for the mentally and physically handicapped. When the Massachusetts Psychiatric Association was formed in 1924 he was naturally elected its President, and shortly before his death he presided at its first scientific session.

In Dr. Fernald there passed away a great citizen, a public servant of the highest standards, a very perfect gentleman. *Si monumentum requiris circumspice.* To find his monument one will not look round any imposing structure, one will not altogether find it in the buildings at Waverley which were his creation; one will find it dispersed through the breadth of the Commonwealth in the simple contented lives of countless men and women, who, but for his training and kindly supervision, would have had a bitter experience of human life.

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CORRESPONDENCE

LONDON LETTER

(From Our Own Correspondent)

MINISTRY OF HEALTH AND PREVENTIBLE DISEASE

A memorandum has been issued recently by the Ministry of Health, written by Sir George Newman, Chief Medical Officer of the Ministry and the Board of Education. It is a notable document from several points of view, and perhaps especially so from its literary style and its lucidity. It sets forth important facts with regard to health in a highly interesting manner and makes valuable suggestions for the prevention of disease and disability.

Indeed, the whole tenor of the memorandum is prevention, and it is urged that in order that preventive measures may be carried out effectively public opinion shall be educated to understand them and take an intelligent interest in them. It is pointed out that public opinion has not kept pace with the improvements and developments in sanitary and health services. Progress has been made in this direction, while changes in fashion and taste have also had their effects. More sensible modes of dressing, the disappearance of tight clothing and trailing skirts, a much larger amount of outdoor exercise, more active amusement and athletics have brought about a vast improvement in the health of girls and women. By these means chlorosis, previously common, and many of the Victorian complaints of women have been practically abolished and the whole standard of their health has been beneficially affected. These remarks as far as women are concerned apply to the upper and middle classes, so called. The girls and women of the poorer classes live much in the same way as they have lived for some considerable time, working at home or in factories and offices. Their lot has been improved physically in that there has been a distinct rise in the standard of cleanliness (speaking generally), personal, domestic and in the factories.

But although public opinion has become more enlightened than formerly, there is still need for further illumination and education on the question of the potentialities and possibilities of preventive medicine as applied to the individual. Daily and in every direction prevention of disease is becoming more of a personal matter, and it is neglect of this personal aspect which is now keeping back and restricting the work and results of preventive medicine. All signs accentuate the need for more public education. Figures are given in the memorandum which prove to the hilt that many deaths and an immense mass of invalidity and disability might be averted if the public understood how preventive measures could be best applied.

It will be impossible within the scope of a letter to deal with all the points discussed in the memorandum and it must suffice to discuss briefly the most arresting.

A considerable amount of emphasis is laid upon digestive disturbances as causes of sickness, and it is noted that knowledge of food values and the needs of the body is lacking, and because of this much money is expended on unsuitable food, while many valuable and cheap foods are neglected entirely. Individuals must be taught what, when and how to cook and eat, and how to protect their food from contamination and keep it in proper condition. Moreover, the diet should be, as far as is possible, sufficient, varied, nutritive and digestible. Meals should not be eaten hurriedly, but should be taken regularly and should be restful occasions. Overeating, overdrinking, and especially drinking alcohol to excess and swallowing food without adequate mastication and deglutition, should be avoided. Education on the use and control of food is, according to Sir George Newman, more urgently needed in England than education on any other health matter.

A subject very near to the heart of Sir George, and one on which he has spoken and written eloquently many times, is that of mother and infant welfare. In England and Wales every year there are lost by death 3000 mothers in childbirth, and many more young mothers unnecessarily injured or invalided thereby. Much of this maternal mortality and sickness could be prevented, yet only a small proportion of the 700,000 mothers who give birth to children every year submit themselves to ante-natal supervision. In 1923 there died in England and Wales 52,582 infants, more than 32,000 of them in the first three months from diseases in a large measure preventible. This means also that many of the survivors of the 700,000 children born every year are impaired or injured and thus handicapped. The main influence which contributes to this loss is motherhood. Since the establishment of schools for mothers, of maternity and infant welfare centers, and of other educational agencies for helping mothers in their maternal duty, the infant mortality rate in this country has fallen by 50 per cent.

Sir George ends a remarkably effective review of the existing situation as regards health affairs in England and Wales, after paying tribute to the medical inspection of school children, by pleading for universal medical inspection, which, he contends, if carried into effect and acted upon wisely, will do more to prolong life, increase its capacity and enhance its value than by the adoption of any other single method or procedure yet known to man.

THE ROYAL BETHLEHEM HOSPITAL

The Royal Bethlehem Hospital for the Insane, commonly called Bedlam, one of the oldest and perhaps the most important mental institution in England, is to be removed from its present situation in Lambeth, on the south side of the Thames, to a more suitable site.

It was first founded in early Norman times. The history of the treatment of the insane through the ages is grim reading, from the days when the monastic view of madness as identical with demoniacal possession established a tradition of ill-treatment which lasted to well into last century. For the greater part of that time scourging and torture were the lot of the insane. To recall Pepys's description of "several poor, miserable creatures in chains, one of them was mad with making verses." Nor did they even escape the stake, in an age when burning was an approved method of driving out the devil.

Bedlam itself was condemned by a committee of the House of Commons in 1815. A few names of the unhappy inmates are still remembered. There was the porter of Oliver Cromwell; there was Nat Lee, the dramatic poet; and Richard Stafford, who had "dispersed many scandalous pamphlets and libels rife with enthusiasm and sedition"; and some demented folk who tried to kill George II, as well as Jonathan Martin, who set fire to York Minster.

It is only in recent years that mental treatment has been based on sound diagnosis, or as sound as may be, and reforms are still called for, in all parts of the world, in the present system of differentiating and treating the insane accordingly.

JUBILEE OF WOMEN'S MEDICAL SCHOOL

The jubilee of the Women's Medical School in London is about to take place. In 1874, when this school of medicine was started, the Medical Register contained the names of two women, Dr. Elizabeth Blackwell, who practised most of her life in America and died there a few years ago. and Dr. Garrett Anderson, and there were fourteen students at the school. Today some 2000 women figure in the British Medical Register, over 1100 of whom have qualified through the school, while 300 students are now enrolled in it and its hospital, the Royal Free.

The original school was in Hunter Street, Bloomsbury, and it is in the same situation—historic, by the way, as one of the Charles was connected in some way with the site—that the present-day beautiful buildings stand. This year Lady Barrett, who as Dr. Florence Wiley held an important post at the school, was elected president of the International Association of Medical Women. Dr. Sophia Jex-Blake was the founder of the London School of Medicine.

In honor of the jubilee three new Chairs are to be founded, in Physiology, Pathology, and Anatomy, bearing the names of the three pioneers, Elizabeth Blackwell, Elizabeth Garrett Anderson and Sophia Jex-Blake. For the maintenance and endowment of these £50,000 must be raised.

NEW BOOKS

William Heinemann (Medical Books, Ltd.) has published recently a book by Mr. J. E. R. McDonagh, F.R.C.S., Surgeon London Lock Hospital. According to the thesis propounded here, disease is simply a successful attempt on the part of the invading organism to rob the protective particles of the blood of their free electricity, and recovery from disease is a reversal of this process, due to the protective particles finally overcoming the invading organism by recapturing this electricity. The modern view of the chemo-therapeutic action of drugs is that they act directly on the body's protective mechanism. If this be stimulated the invader is routed; if it be destroyed the invader gains the day. A drug does not act directly upon the disease but urges protective mechanism to defend the body as effectively as in it lies. This is a well-written work, alive with original conceptions, and is one to be read carefully. Mr. McDonagh's views are revolutionary but stimulating.

CORRECTION OF A MISLEADING IMPLICATION

Massachusetts Department of Public Health,

Editor, Boston Medical and Surgical Journal:

Will you kindly give space in the JOURNAL for the correction of a misleading implication which occurred during the discussion at the meeting of the Section of Obstetrics and Gynaecology in June concerning the study of material mortality this Department is now carrying on, as reported on page 633 of the issue of the JOURNAL of October 2, 1924.

It is here stated by one of the speakers as quoted that this study is being carried on by "doctors," the quotation mark and further comment indicating clearly that there was doubt in the mind of the speaker as to the fitness of these investigators to interpret the material they are collecting.

I should like to say for the better understanding of your readers that each one of the physicians doing this work holds a degree in medicine respectively from Johns Hopkins, Ann Arbor and Boston University Schools of Medicine. All have taken postgraduate work as well, besides having had technical experience fitting them for the work they are undertaking. One has also done work in the Harvard School of Public Health.

Appreciating the courtesy I feel confident you will extend in allowing me this opportunity to correct an impression unjust alike to the individuals concerned and to the Department which I represent, I am

Yours very truly,
EUGENE R. KELLEY,
Commissioner of Public Health.

MEDICAL SOCIETY OF VIRGINIA

Dr. W. L. Burrage,
182 Walnut Street, Brookline 46, Mass.

Dear Sir:

I had planned to attend the meeting of the Secretaries in Chicago this fall, but find now that I cannot be there.

You doubtless recall the interest I have taken in the past four years in securing a reduction in the cost of automobile and physicians' malpractice insurance.

With the assistance of several local business men, including experienced insurance men, the following plan has been worked out and is herewith submitted to the individual secretaries of the State Medical Society for their coöperation.

1. It is proposed to organize a mutual liability insurance company, writing automobile liability, malpractice and accident insurance.
2. This organization will be entirely separate and distinct from the American Medical Association and the State Medical Societies and will, in no way, involve itself in the affairs of these organizations.
3. Its business will be limited strictly to members of the State Medical Society which make up the American Medical Association.
4. Where desirable the business of the company will be written in the offices in which the business of the State Society is carried on; also, if desirable, the State secretary will act as general agent for the company. His compensation for this additional work will be based on the volume of business done.
5. In order for a mutual company to transact business throughout America it is necessary that it have resources to the amount of $250,000. This money may be borrowed from members, bonds of the company being issued to the members and payment of as much as seven per cent. on the loan is legal. It is proposed, therefore, that we locate members who will be willing to put up $250.00 to $1000.00 each to make up these required assets. The loan will be returned as rapidly as the company's premium income can replace the amount borrowed.
6. It is estimated that the physicians of America pay three million a year for this insurance and that after the first year this amount can be reduced to one and a half million dollars.

The writer has secured the promise of more than sufficient funds to make up Virginia's pro rata share of the loan and the coöperation of the secretaries is requested, or if preferable secretaries are requested to give the writer names of the physicians in their State who might be induced to help in this undertaking.

The following are suggested for the first board of trustees: Dr. Allen H. Bunce, Atlanta, Ga.; Dr. C. N. Combs of Indiana, Dr. T. B. Throckmorten of Iowa. Dr. E. L. Hunt of New York, Dr. Holman Taylor of Texas, Dr. D. K. Martin of Ohio, together with six or eight experienced business and insurance men and the writer.

Your prompt reply to this will be greatly appreciated.

Yours truly,
G. H. Winfrey,
Secretary-Treasurer and Business Manager.

November 28, 1924.

Mr. G. H. Winfrey,
Secretary-Treasurer and Business Manager,
Medical Society of Virginia.

Dear Sir:

Answering your letter of November 19, sent to me by the kindness of Dr. Olin West, in whose care it was directed, the whole subject of insurance was threshed out by the Massachusetts Medical Society in the fall of 1921. I am sending you a copy of the proceedings of our Council in November of that year that you may see how the subject was treated at that time. Under the charter our society may not engage in commercial insurance. It has been thought inexpedient to have the charter changed. We offer malpractice defence to all Fellows in good standing. Your letter I am sending to the editor of our official organ, the Boston Medical and Surgical Journal, for whatever publicity the editor of that publication sees fit to give it.

Very truly yours,
W. L. Burrage, *Secretary.*

A CLINIC FOR SURGICAL EXAMINATIONS

Editor, Boston Medical and Surgical Journal:

During the winter, 1923-24, it was announced that Boston University School of Medicine was about to establish a clinic for surgical examinations at the Boston Y. M. C. A. This clinic has now been in operation almost a year and it is felt that perhaps some word of explanation would be in order at this time.

All members of the Y. M. C. A. are eligible for this examination, which may be obtained by making proper appointment. Examination is compulsory for those men living in the dormitory. These is an examining physician in charge, who is assisted by two senior medical students from Boston University School of Medicine. It should be understood that the service is in no sense a medical clinic and that medical treatment is not undertaken.

So far the objectives have been:

1. To discover errors in physical development, posture, habits of living, etc., that are correctible or that may be helped by the advice of the examiner, supplemented by corrective methods supplied by the physical director.
2. To discover by history and examination evidence of conditions which may warrant further investigation or medical and surgical treatment. An attempt is made to put each case in contact with his physician and to report to the physician the findings. In certain cases physical activities at the Y. M. C. A. are discontinued until the report is received from the physician.
3. The training of advanced medical students in the examination of a large number of men who are apparently well.
4. The thoroughness of the examination and the knowledge of the cases which are benefited thereby should create a desire on the part of the candidates for periodic health examinations by their physicians. It is pointed out, however, that they will probably not be satisfied with the casual inspection so often given in the past.

There may have been a misapprehension on the part of many physicians in regard to the work this examining clinic is trying to do. There is no intent to encroach upon the domain of the family physician, but rather to seek for coöperation and to see to it that all necessary information obtained at the time of the examination is placed in his hands.

A follow-up system is now being established in the hope that information may be obtained which will indicate what proportion of the men have actually carried out the suggestions made and what benefits have accrued.

Physicians who are interested in the movement are cordially invited to visit the clinic. Information as to hours, etc., may be obtained from the Y. M. C. A. secretary.

Yours sincerely,
Nathan H. Garrick,
Examining Physician.

ACUTE EPICRANIAL PARESTHESIA

Editor, Boston Medical and Surgical Journal:

A syndrome, apparently not well known and perhaps new, has recently been noted in relation to common colds or slight attacks of grippe. Three cases were seen in one day at the Massachusetts General Hospital, and within the last two weeks perhaps a dozen patients have complained of the annoying symptoms. I am told that the same, or a very similar syndrome, has been reported elsewhere in the United States.

The symptoms are largely sensory and subjective. The paresthesia is usually preceded by a common cold, without a marked rise in temperature, or mild grippe associated with malaise and myalgia. Within a few days paresthesia of the scalp or face is noticed; in all cases seen the symptoms have been unilateral. The sensation, often of the scalp only, is described as burning, tearing, "as if the flesh was denuded and raw, being particularly sensitive to a breath of air," or "like electricity." There is no paroxysmal pain, as in trigeminal neuralgia, but the discomfort may be intense. Brushing the hair becomes extremely painful, the least movement of the hair being almost unbearable. Male patients find great difficulty in shaving the affected side. The paresthesia is also described as "throbbing"; in one patient this sensation prevented sleep for a number of nights. The discomfort is confined to the skin, the deeper structures, bone, teeth, and mucous membranes, not being affected. No hepetic eruptions have been noted.

The scalp is more commonly involved than the face. The paresthesia may last from a few days to three weeks, gradually decreasing in severity. Occasionally the suboccipital area, supplied by the upper cervical nerves, is involved. Tinnitus may be a second symptom, appearing on the same side as the trigeminal paresthesia. It is often persistently annoying, but disappears with the other symptoms. No deafness is noted by the patient. Motor paralysis of the other cranial nerves has not been found. Except for some general fatigue the patients are usually free from other symptoms.

No definite etiology is known. The symptoms would point to toxic irritation of the trigeminal and auditory nerves, associated with mild upper respiratory tract infection, perhaps with the influenza group of bacteria.

Treatment is symptomatic and general. Most patients feel better out of bed, or even in the open air.

In one patient the "throbbing sensation" of the scalp and tinnitus was increased by lying down. Pyramidon in five-grain doses may be tried to relieve the paresthesia, and luminal, grains one and one-half, the tinnitus. Gentle massage of the scalp followed by a warm water and oil shampoo brought relief to one patient. No drugs are known which shorten the duration of the syndrome or markedly lessen its intensity.

Henry R. Viets, M.D.

AMERICAN MEDICAL ASSOCIATION

Council on Pharmacy and Chemistry

Editor, Boston Medical and Surgical Journal:

In addition to the articles enumerated in our letter of October 28 the following have been accepted:

Hoffmann-LaRoche Chemical Works—
Secacornin
Thigenol

Intarvin Co., Inc.—
Intarvin

Eli Lilly & Co.—
Ampules Ouabain, 0.0005 Gm. (1/128 grain)—Lilly
Hypodermic Tablets Strophanthin 1/100 grain—Lilly
Hypodermic Tablets Strophanthin 1/120 grain—Lilly
Iletin (Insulin—Lilly) U-80

Merck & Co.—
Benzyl Succinate—Merck

Parke, Davis & Co.—
Ampoules Adrenalin Chloride Solution Rx 1, 1:10000, 1 c.c.
Ampoules Adrenalin Chloride Solution Rx 2, 1:2600, 1 c.c.
Ampoules Adrenalin Chloride Solution, 1:1000, 1 c.c.

Sharp & Dohme—
Hypodermic Tablets Strophanthin (1/200 grain)—S. & D.
Ergotole:
Ampules Ergot, 1 c.c.
E. R. Squibb & Sons—
Insulin—Squibb, 10 Units
Insulin—Squibb, 20 Units
Swan-Myers Co.—
Sterile Ampoules Mercuric Potassium Iodide, 0.017 gm. (¼ grain)—Swan-Myers

Synthetic Drug Co., Inc.—
Compressible Capsules Mercury Salicylate "Synthetic"
1 grain for Intramuscular Injection
Compressible Capsules Mercury Salicylate "Synthetic"
1½ grains for Intramuscular Injection
Compressible Capsules Mercury Salicylate "Synthetic"
2 grains for Intramuscular Injection

Winthrop Chemical Co.—
Novasurol
Novasurol Ampules

Yours truly,
W. A. Puckner, *Secretary,*
Council on Pharmacy and Chemistry.

DANGER FROM ZINC STEARATE DUSTING POWDERS

Editor, Boston Medical and Surgical Journal:

The readers of Dr. O'Keefe's article on Zinc Stearate Insufflation in Infancy, in your issue of October 16, are hardly likely to grasp the gravity of the danger from zinc stearate dusting powders. It may be well, therefore, to let them know that the Committee on Zinc Stearate Dusting Powders, appointed by the Board of Trustees of the American Medical Association, has collected reports of 129 accidents from such powders, of which 27 proved fatal.

The committee named was created to carry into effect a resolution of the House of Delegates of the American Medical Association, adopted at San Francisco in June, 1923, calling attention to the situation and directing the Board of Trustees to promote measures of safety. (*J. A. M. A.*, July 14, 1923, p. 121.) At the meeting of the House of Delegates in Chicago in June last, the first report of the committee was submitted. (*J. A. M. A.*, July 12, 1924, pp. 120-121.) The House thereupon authorized the Board of Trustees to continue its activities and to seek protective legislation if they deemed it necessary.

The matter of safeguarding the distribution of stearate of zinc dusting powders has been taken up with various packers and jobbers, some of whom had even before the appointment of the committee adopted precautionary measures, such as self-closing cans and warning labels. A conference with representatives of the trade has been called for October 31, to discuss the situation and to determine what course to pursue.

The committee will be glad to have reports of such further cases as come to the knowledge of the profession sent to the committee at the headquarters of the American Medical Association, 535 North Dearborn Street, Chicago, Illinois.

Yours truly,
Isaac A. Abt, *Chairman,*
Committee on Zinc Stearate Dusting Powders.

HEALTH EXAMINATIONS

Worcester, Mass., November 9, 1924.

Editor, Boston Medical and Surgical Journal:

As one who is very much interested in public health work, I wish to compliment Dr. Lee and his Committee on Health Examinations for the excellent report printed in the November 6 issue of the Journal.

For several years I have been urging my patients to have a yearly examination, and from that rather limited experience I would like to make a few suggestions.

I would like to adopt their blank as published, with a few additions, namely:

1. Enlarge the history blank by asking for more detail in regard to dietetic habits. It is almost axiomatic that faulty dietetic habits are the cause of a great deal of chronic disease.
2. Enlarge the history blank still farther by asking for more detail in regard to individual habits of exercise.
3. In the examination blank add a space for temperature record, recent gain or loss in weight, girth of chest and abdomen, and result of rectal or prostate examination.

I would like to suggest also that the committee prepare a small folder, with cuts if possible, for the office table and for mailing to patients with the monthly bills, which would give in a simple but convincing way the benefits of the annual examination. Possibly this might find its way into the monthly bulletins of the Chamber of Commerce and other semi-social or fraternal organizations and in that

way get a widespread publicity of this modern method **of life extension.**

Further I would suggest our medical schools, in **their extension courses, give** a few lectures and clin**ical demonstrations** to the general practitioner how **best to make** this examination, and thus stimulate **him to look** for minor deviations from the normal **and make him** more keen in his discovery of the very **beginnings of what** may be disease.

I hope that this report will be accepted and the **committee instructed** to continue its study of the **subject.**

Yours truly,
Roy J. Ward.

SCARLATINAL ANTITOXIN AND ANTI-SCARLATINAL SERUM

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

Editor, Boston Medical and Surgical Journal:
126 Massachusetts Avenue, Boston, Mass.

Dear Mr. Editor:

The recent studies on scarlet fever by Dochez, the Dicks, Zingher and others have aroused a new and eager interest in ways for the prevention and treatment of this disease. This interest has resulted in numerous inquiries to this Department as to our opinion concerning the value of the Dick test, active immunization with Dick toxin and the use of the scarlatinal antitoxin of the Dicks and the anti-scarlatinal serum of Dochez. Furthermore, there has arisen a demand for these new biologic agents and the question has been frequently put whether this Department would prepare and distribute these products in the near future. It therefore seems desirable at this time to issue a statement concerning the Department's policy in regard to this important subject.

We feel that the studies of Dochez and the Dicks may be regarded as most valuable and significant contributions to our knowledge of the etiology of scarlet fever, and if substantiated by further study they may prove to be the means for controlling the disease. We feel, however, that these measures are still in, and should for some time be held to be in, the investigative stage. From a theoretical standpoint we need further confirmation of the specific etiological role of streptococci in scarlet fever; from both a theoretical and practical standpoint we need further evidence that the so-called toxin produced by scarlatinal streptococci is the specific poison of scarlet fever; that the Dick test is an accurate and reliable means for testing susceptibility or immunity to scarlet fever; that Dick toxin induces active immunity to the disease; that the Dick antitoxin and the anti-scarlatinal serum of Dochez are curative agents of value.

The available reports on the use of these procedures and agents, promising as they appear to be, are still meagre. They also reveal certain inconsistencies and present questions that cannot now be answered. The United States Hygienic Laboratory, recognizing the difficulties presented in the standardization of the toxin and serums, has not yet issued any licenses for their manufacture.

Until we have further confirmation of the accuracy and dependability of the test, more clinical data on the protective action of toxin injections and the curative effect of the serums, and until the preparation of these products is standardized by Federal regulation, this Department, following its established policy of conservatism, feels that the time has not yet come either to advocate the use of these procedures and agents in medical or public health practice or to supply these products for general distribution.

At the Antitoxin and Vaccine Laboratory this Department is planning on an experimental scale the production of anti-scarlatinal serum by the Dochez method with the advice and coöperation of Doctor Dochez; but for the present the production of Dick toxin and antitoxin will not be undertaken.

Very truly yours,
Eugene R. Kelley,
Commissioner of Public Health.

CONNECTICUT DEPARTMENT OF HEALTH

Weekly Morbidity Report for the Week Ending December 13, 1924

(Including all cases reported before 11 A. M., Monday, December 15, 1924)

Diphtheria

Fairfield County	
Bridgeport	2
Fairfield	1
Norwalk	4
Stamford (C)	2
Stratford	1
Westport	1
Hartford County	
Avon	1
Enfield	3
Hartford	5
New Britain	6
West Hartford	1
Litchfield County	
Torrington (B)	2*
Watertown	1
Middlesex County	
East Hampton	1
New Haven County	
Ansonia	1
Beacon Falls	3
New Haven	2
Wallingford (B)	2
Waterbury	10
New London County	
Groton (B)	1
Norwich (C)	1
Tolland County	
Mansfield	1
	—
State total	52
Last week	74

The following diphtheria bacilli carriers were reported:

East Haven	1
Hartford	6
Milford	4
New Haven	1
Norwalk	23
Simsbury	1
Waterbury	1
West Hartford	1

Typhoid Fever

Fairfield County	
Greenwich	1
Norwalk	1
Westport	1
Hartford County	
New Britain	1
New Haven County	
Meriden (C)	1
New Haven	1
	—
State total	6
Last week	5

Scarlet Fever

Fairfield County	
Bridgeport	11
Danbury (C)	3
Greenwich	1
Stamford (C)	3
Stamford (T)	2
Stratford	1
Hartford County	
Avon	1
Bristol	4
Hartford	9
Manchester	3
New Britain	30
Newington	5
Plainville	2
West Hartford	1
Wethersfield	1
Litchfield County	
Litchfield	5
Thomaston	8
Torrington	5*
Watertown	1
Middlesex County	
Cromwell	5
Middletown (C)	2
Middletown (T)	2
Portland	2
New Haven County	
Beacon Falls	1
East Haven	2
Hamden	2
Madison	3
Meriden (C)	1
Middlebury	2
New Haven	38
Waterbury	4
West Haven	1
New London County	
Groton (B)	3
Groton (T)	1
Lebanon	1
New London	1
Norwich (T)	5
Preston	2
Tolland County	
Mansfield	1
Windham County	
Pomfret	1
	—
State total	176
Last week	145

Measles

Fairfield County	
Ridgefield	1
Hartford County	
Hartford	1
New Haven County	
New Haven	8
Waterbury	1
New London County	
Griswold	1
	—
State total	12
Last week	6

Whooping Cough	
Fairfield County	
Greenwich	2
Stamford (C)	7
Hartford County	
Enfield	8
Hartford	2
Manchester	2
Litchfield County	
Plymouth	1
New Haven County	
Hamden	3
Meriden (C)	2
New Haven	13
Waterbury	2
New London County	
Groton (B)	11
New London	2
State total	55
Last week	46

Other Communicable Diseases	
Cerebrospinal men.	1
Chickenpox	78
Conjunctivitis inf.	1
Favus	1
German measles	30
Hookworm infection	1
Influenza	2
Mumps	22
Pneumonia (lobar)	31
Poliomyelitis	1
Septic sore throat	19
Tuberculosis (pul.)	36
" (other forms)	2
Gonorrhea	26
Syphilis	20

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

Diseases Reported for the Week Ending December 20, 1924

Diseases	*No. of Cases*	*Diseases*	*No. of Cases*
Anterior poliomyelitis	1	Ophthalmia neonatorum	22
Anthrax	1	Pneumonia, lobar	110
Chickenpox	301	Scarlet fever	313
Diphtheria	153	Septic sore throat	2
Dog-bite requiring anti-rabic treatment	4	Syphilis	34
Encephalitis lethargica	6	Suppurative conjunctivitis	16
German measles	45	Tuberculosis, pulmonary	106
Gonorrhea	92	Tuberculosis, other forms	11
Influenza	11	Typhoid fever	16
Measles	141	Whooping cough	71
Mumps	121		

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

Diseases Reported for the Week Ending December 27, 1924

Diseases	*No. of Cases*	*Diseases*	*No. of Cases*
Chickenpox	155	Pneumonia, lobar	66
Diphtheria	111	Scarlet fever	282
Encephalitis lethargica	7	Septic sore throat	3
Epidemic cerebrospinal meningitis	1	Syphilis	47
German measles	47	Tetanus	1
Gonorrhea	70	Suppurative conjunctivitis	18
Influenza	17	Tuberculosis, pulmonary	46
Measles	112	Tuberculosis, other forms	20
Mumps	48	Typhoid	12
Ophthalmia neonatorum	37	Whooping cough	32

REPORTS AND NOTICES OF MEETINGS

A physiological conference will be held Wednesday, January 14, at the Bowditch Library, Building C of the Harvard Medical School, at 4 P. M. Dr. H. E. Himwich will speak on "The Role of Fat in Muscular Exercise."

HARVARD MEDICAL SOCIETY

The next regular meeting of the Harvard Medical Society will be held as usual in the amphitheatre of the Peter Bent Brigham Hospital, Jan. 13, 1925, at 8:15 P. M. The program follows:

1. Demonstration of cases.
2. Some remarks on students' health. Dr. Roger I. Lee.
3. Fame on a biscuit. Dr. Harvey Cushing.

All members of the medical profession, medical students and nurses are invited.

Very sincerely yours,
Dr. S. A. Levine, *Secretary.*

THE NEW ENGLAND WOMEN'S MEDICAL SOCIETY

The Annual Dinner will be held at the Hotel Victoria, Dartmouth Street, Boston, Thursday, January 15th, at 6 P. M.

Miss Charlotte G. MacEwan, Instructor in Dancing in the Department of Hygiene and Physical Education of Wellesley College, will speak on "The Significance of Dancing in Therapeutics." There will be demonstrations by her pupils.

Miss Margaret Johnson of the same Department at Wellesley will play the piano for the dancing.

Anna Quincy Churchill, M.D.,
Secretary.

NEW ENGLAND PEDIATRIC SOCIETY

The eighty-eighth meeting of the New England Pediatric Society will be held at the Boston Medical Library on Friday, January 9, 1925, at 8.15 P. M.

The following papers will be read:

President's Address, Kenneth D. Blackfan, M.D., Boston, Mass.; Cranial Rickets, Henry E. Utter, M.D., Providence, R. I.; Some Clinical Aspects in Fifty Cases of Tetany, Robert M. Lord, M.D., Providence, R. I.

Light refreshments will be served after the meeting.

Kenneth D. Blackfan, M.D., *President.*
Joseph Garland, M.D., *Secretary.*

BOSTON ASSOCIATION FOR THE PREVENTION AND RELIEF OF HEART DISEASE

Meeting at the amphitheatre of the Peter Bent Brigham Hospital, Thursday, January 15, 1925, at 8:15 P. M.

Subject:—"All Heart Symptoms are not Serious," Dr. Henry Jackson.

Doctors, students and all others interested are invited.

S. A. Levine, *Secretary.*

GREATER BOSTON MEDICAL SOCIETY

Ear, Nose, and Throat Night, Monday,

January 12, 1925

Boston Medical Library, John Ware Hall,
8 The Fenway, Boston

Business meeting, 8:15 to 8:45 p. m. Program 8:45 p. m.:

1. Louis M. Friedman, M.D.
"Mastoid Infections; Diagnosis and Operative Treatment" 20 minutes
2. Louis Arkin, M.D.
"Tonsillectomy and Adenotomy. A study of 1000 cases"
3. Harry P. Finck, M.D.
"Foreign Bodies in the Respiratory Passages and Oesophagus" 20 minutes

Discussion opened by Drs. Philip E. Meltzer, Louis E. Wolfson, Samuel Cline, and Joseph Prenn.

Physicians and medical students are cordially invited to attend this meeting.

LOUIS J. ULLIAN, M.D. *Secretary.*

ESSEX SOUTH MEDICAL SOCIETY

THE next meeting of the society will be held January 14th at Boston City Club. The eminent professor of Public Health at Yale University, Dr. C. E. A. Winslow, will address the meeting on the subject of Industrial Tuberculosis.

It is hoped that a large attendance will be there to greet Professor Winslow.

Any physician interested in the subject is cordially invited to attend.

Dinner will be served at 6:30 P. M., in the large banquet hall. A cover charge, as yet undetermined, will be made.

Any comments on your part as to the importance of this subject will be greatly appreciated.

J. ARMAND BEDARD,
President.

BOSTON CITY HOSPITAL

A STAFF clinical meeting will be held in the Cheever Surgical Amphitheatre on Friday, January 9, at 8.15 P. M. Subject: Early and Late Trauma—Its Treatment and Prognosis—by Dr. F. J. Cotton and Dr. J. H. Shortell. Open discussion. Physicians, medical students and nurses invited. Refreshments.

JOHN J. DOWLING,
Superintendent.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

THE January Meeting for Medical Improvement has been postponed to the second Thursday, the first Thursday being a holiday. The meeting will be at Norfolk County Hospital, South Braintree, Thursday, January 8, 1924, 12 noon. Speaker: Dr. John T. Bottomley. Subject: "Acute Indigestion," so called. Discussion.

MEDICAL VETERANS' CLUB

THERE was a meeting of the Lawrence Medical Veterans of the World War Club at the Methuen Tavern, Methuen, Mass., Thursday, Dec. 18, 1924, at 8 P. M.

THE MEMORIAL HOSPITAL, WORCESTER, MASS.

THE trustees of the Memorial Hospital announce the opening of an out-patient clinic exclusively for the treatment of scoliosis.

Reliable statistics show that the life expectancy of these cases is greatly shortened under inadequate treatment. The results of recently devised methods are so remarkable that the hospital feels impelled to add to its present activities a special clinic for this work.

The clinics will be held Wednesdays and Saturdays at ten o'clock A. M. in charge of Dr. Charles E. Ayers.

SOCIETY MEETINGS

Essex North District Medical Society

May 6, 1925. Annual meeting at Lawrence.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held on the second Tuesday of January, March and May.

Hampden District Medical Society

Meetings to be held on the third Tuesday of January and the third Tuesday in April.

Hampshire District Medical Society

The meetings will be held the second Wednesday of November, January, March and May.

Middlesex East District Medical Society

Wednesday, January 21. Harvard Club. Dr. Franklin K. White, "Diagnosis of Gall-Bladder Disease."

Wednesday, March 18. Harvard Club. Dr. John H. Cunningham, "Urinary Retention: Its Significance and Treatment."

Wednesday, April 15. Harvard Club.

Wednesday, May 13. Colonial Inn, North Reading.

Middlesex North District Medical Society

January 28, 1925.
April 29, 1925.

Middlesex South District Medical Society

Winter Schedule—The plans for winter meetings of the Society include the stated meetings in October and April, two hospital meetings, and five meetings to be held in conjunction with the Suffolk District Medical Society and the Boston Medical Library (two surgical, two medical, and one general).

Norfolk District Medical Society

January 27, 1925. Masonic Temple. Subject: "Some Trends of Medical Teaching and Medical Practice." Speakers: Drs. A. S. Begg and W. P. Bowers.

February 24, 1925. Masonic Temple. Subject: "The Need of Periodical Physical Examinations and How to Make Them." Speaker: Dr. Francis H. McCrudden. A second speaker will be selected to present another subject at this meeting.

March 31, 1925. Tufts College Medical School. This meeting given over to Drs. Leary and Watters for the purpose of giving us a medical examiners' talk.

Norfolk South District Medical Society

Meetings will be held the first Thursday of each month from October to May, inclusive, at 12 noon, at the Norfolk County Hospital, South Braintree.

Suffolk District Medical Society

January 28. General meeting, in association with the Boston Medical Library and the Middlesex South District Medical Society. "Some Experiences of a Medico-legal Pathologist" (lantern slides), Dr. George B. Magrath.

February 25. Surgical Section, in association with the Middlesex South District Medical Society. "Pyelonephritis," Dr. Arthur H. Crosbie.

March 25. Medical Section, in association with the Middlesex South District Medical Society. "The Treatment of Pneumonia," Dr. Edwin A. Locke.

April 29. Annual meeting. "Hypertension and Longevity," Dr. Harold M. Frost.

Worcester District Medical Society

February 11, 1925. Memorial Hospital, Worcester. Papers will be read by the members of the hospital staff.

March 11, 1925. St. Vincent's Hospital, Worcester. Papers will be read by the members of the hospital staff.

April 9, 1925. Subject and speaker to be announced.

May 14, 1925. Annual meeting.